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ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

STUDIES OF MASTOID DISEASE BY THE X-RAYS WITH CLINICAL OBSERVATIONS AND OPERATIVE FINDINGS.*

DR. MATTHEW S. ERSNER, Philadelphia, Pa.

In preparing this paper I have tried to present the subject in as practical a form as possible and this alone, I hope, will suffice to convey a crystalized idea of the subject.

I have studied in conjunction with Dr. Pfahler 142 mastoid cases.

In this little bundle of records there is a store of knowledge which in quiet moments I often review. The X-ray is a study of dead shadows of varying densities, which to the casual onlooker means nothing, but to the donors and clinicians the plates recall trying moments, aching pains and sleepless nights, etc. So when I look at the plates there appears through each, the hazy image of the patient. From our collection, we have selected 19 slides representing various typical and atypical interesting acute mastoid types. It is our routine to order X-ray examinations for every mastoid patient, whether private, semi-private or ward, irrespective of the clinical diagnosis, whether conclusive or not. Some think this procedure extravagant, others forget the X-ray when the clinical find-

*Presented by invitation in conjunction with X-ray studies by G. E. Pfahler. Illustrated by lantern slides before Section on Otology and Laryngology, of the College of Physicians of Philadelphia, November 21, 1923.

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ings point toward operation. I too was skeptical at first in my inclination towards the X-ray in mastoid disease, but have since learned its indispensibility.

Many men usually X-ray their atypical cases only, which under such circumstances we may have atypical anatomical abnormalities to deal with and the shadows may therefore be misunderstood. The X-ray is the sequence of a physical, mechanical and chemical procedure, accomplished by the penetration of the rays through the suspicious area and registering their course by casting various shadows. I believe that every shadow is a message for us to interpret. These may be helpful, misleading or misunderstood. It should be our aim in life to have the most knowledge in the field of our daily pursuit and since knowledge is based upon experience accumulated, one should therefore in this instance study as many plates as possible and correlate the various shadows with the clinical and operative findings. The X-ray plates to me are less puzzling daily, and I feel that I am having less difficulty in interpreting them. I often see shadows which recall cases similar in nature which heretofore were Chinese puzzles to me but now are concrete facts without pitfalls.

What should we as clinicians expect from the X-ray in mastoid diseases.

1. Comparative study between the normal and the diseased mastoid. (Both mastoids are usually developed alike and any deviation from the normal will be divulged by the X-ray plate.)

2. Type of mastoid, whether pneumatic, non-cellular or sclerotic.

3. Topographical and regional anatomy of the mastoid, its cellular distribution, type of cells, position of the sinus and its surrounding area.

4. In atypical mastoiditis the X-ray is indispensible as an aid in diagnosis.

5. Cavity formation.

6. Prognosis is often made by consecutive X-ray studies. (A mastoid with large cells may undergo resolution without operative interference as the drainage in such mastoid is not impeded as frequently as in the other mastoid types.

7. Diagnosis of peri-sinus abscess.

8. Epidural abscess diagnosed.

9. Bilateral suppuration.

10. Sclerotic mastoid.

11. In children where the cells are not fully developed, a Roentgenologic diagnosis may often be made when the antrum is located as there are a few cells invariably present there.

Let us visualize hypothetically step by step the pathological changes taking place in the mastoid and interpret these phenomena on the X-ray plates, providing these are pneumatic mastoids. In an acute mastoid of recent origin we at first have hyperemia and the congestion is the same as in any other part of the body. Roentgenologic studies of both mastoids reveal that the normal side is clear and that the cellular components stand out sharply with distinct cellular septa while in the congested side all the normal mastoid elements are present, but in addition there is a slight haziness appearing as though there were a slight error in focusing.

In this stage either resolution takes place or the pathology continues. We may assume that the bacteria here are active and that the mucous membranes lining the mastoid cells are becoming more edematous. We also have a constant infiltration of round cells and an increase in the leukocytes, the congested area gradually becomes purulent. The X-ray plates at this time will reveal marked cloudiness with a tendency to obliteration of the cellular spaces appearing as though they were filled almost to the brim. The cellular septa are still visible as a minute network of fibrilated strands and our interpretation of the plates at this stage should be exudation present. In the advent of an increase in the purulent constituency there is also an increase in the intra-cellular pressure. The picture now presents a distinct opacity with a blurring of the cellular septa. In case the surgeon decides to follow the conservative course of watchful waiting, the lateral sinus under these conditions should be outlined and studied carefully in the consecutive pictures. When the lateral sinus is lost, we may suspect a peri-sinus abscess with a loss of the bony structure covering the anterior bony sinus wall. Operation under these circumstances is imperative and should be done immediately. Absorption, liquifaction and cavity formation appears on the X-ray plate as a dark spot or as a shadow more or less regularly outlined with the edges blurring and no trabecula visible. In contra-distinction and normal large cell is irregular with the edges sharply outlined, and the cellular septa are distinctly visible. A loss of cellular structures over the zygomatic area with an extension of the haziness towards the squamous portion of the temporal bone is suggestive of an epidural abscess.

In the 19 cases presented here, we have a detailed study including the history, clinical and operative findings, which are correlated with the Roentgen studies.

CASE 1: Baby R., age 18 months. (2-5-23.)

History: Baby is a child of a physician whose mother and brother were sick with grip. As they were recuperating this child fell a victim. The temperature fluctuated for six days when I was called to see the baby and I found a bulging right ear drum. Incision of ear drum with flow of free pus. For four days it looked as though all was well when there was a recrudescence of the symptoms. The ear drum was reincised five times but the pus continued to discharge profusely. A pediatrician was called in during my absence and he incised the other ear drum. The middle ear in this instance became infected but the drainage was not very profuse and about ten swabbings daily sufficed to keep the middle ear clean. After several weeks of bilateral suppuration, the clinical signs and symptoms centered upon the right ear. The X-ray showed either destruction or undeveloped mastoid cells.

Laboratory Findings: Leukocyte count 14,000. Urine analysis negative. There was also a secondary anemia present, poikilocytes, anisocytes and nucleated red blood cells.

X-ray Report: 1. Mastoid unusually large for a child of 18 months. Disease apparently involves the entire area. About 4 cm. in diameter. There are transparent areas in the zygomatic region which are probably also decalcified trabeculae.

On March 6, 1923, I thought I felt a thickening of the periostium and on March 9th there appeared a slight displacement of the auricle.

Operative Findings: 1. Periosteum slightly thickened and not adherent. 2. Cortex thin. 3. Small pin point perforation of cortex opposite the spine of Henle (not developed). 4. Pus under pressure. 5. Small cavity at tip and extensive necrosis of the zygomatic cells. The rest of the mastoid cavity was filled with pus, granulation tissue and in discrete areas, the cellular elements were undisturbed. 6. The mastoid cavity was very large and the antrum very deep.

CASE 2: F. R., age 6 years. (4-9-23.)

History: Patient had measles which was complicated by an acute suppurative otitis media. The ear drum on the right side ruptured spontaneously and when seen there was a profuse discharge. The temperature fluctuated between 99 and 103 daily with severe pain over the right mastoid. The other general symptoms such as restlessness, insomnia accompanied.

Laboratory Examination: Leukocyte count 16,000. Urine negative.

Physical Examination: (a) Profuse aural discharge. (b) Pus under pressure. (c) Sagging of post superior canal wall. (d) Impaired hearing. (e) Loss of post auricular sulci only one-half fold remaining while three and one-half folds were present on the good side. (f) On palpation there was a slight thickening of the periosteum and on deep pressure I located two pronounced tender spots, one at the posterior portion of the tip and the other at the distal end of the zygomatic ridge.

X-ray Report: Mastoid area comparatively small. The mastoids belong to the small pneumatic type. The left side is more fully developed than the right. There is evidence of disease with absorption of lime salts in the regions of the knee of the lateral sinus and of the tip. The sinus walls are well marked.

Operative Findings: 1. Periosteum thickened and stripped with difficulty. 2. Cortex congested with slight greenish discoloration at the tip. 3. Cortex thick. 4. Pus thick and creamy but not under tension. 5. Two cavities were present, one at the tip and the other at the knee of the lateral sinus. 6. The zygomatic cells were partly destroyed but the rest of the mastoid cells were filled with granulation tissue and pus; the cells were soft but not liquified.

CASE 3: Mrs. T., age 45. (4-6-23.)

History: Patient had an acute suppurative otitis media, which was a complication of grip. She was first seen in my office March 24, 1923, complaining of pain in the ear with tinnitus and impaired hearing.

Examination: A bulging ear drum was revealed which I incised and free pus was found. Four days later (3-28-23) she developed pain over the mastoid with temperature ranging around 100° with no abatement of the symptoms. On April 3, 1923 the discharge from the ear became more profuse. She was sent into the hospital for observation where she was X-rayed. Her condition cleared up after being in bed for one week.

X-ray Report: Mastoid is of the large-cell pneumatic type. The left is more extensive than the right. The right cells are clouded throughout.

Conclusion: This mastoid was of the large pneumatic type and by putting the patient to bed, it gave her a chance to fight the infection off and the large cells drained themselves and spontaneous cure resulted.

CASE 4: L. G., age 5½ years. (9-22-23.)

History: Patient had measles complicated by an acute suppurative otitis media. When first seen in the office, she gave a history

of a discharging ear three weeks prior with spontaneous rupture of the ear drum. The discharge lasted for nine days after which the ear became dry. Mother noticed that there was a complete loss of hearing in the right ear and this was verified during conversation. The child's chief complaint was pain at night and pain upon manipulation.

Examination: Child was very intelligent and co-operative. (a) Impaired hearing with complete loss of whispered voice and only five feet of spoken voice. (b) Fork tests: Loss of low tone; Rinne negative; Weber lateralized to the right; Schwabach plus. (c) Ear drum apparently normal. (e) Inspection: Loss of post-auricular sulci while normal side had three folds. (f) Thickening of periosteum over mastoid. (g) Slight suggestion of displacement of auricle. (h) Pain illicited only on deep palpation in the following areas: 1 Over emmisary vein; 2 Over tip; 3 Over antrum.

X-ray Report: Mastoid pneumatic in type, large cells. Local bone absorption at the tip, with general clouding of the remainder of the cells.

Operative Findings: 1. Periosteum slightly thickened but not adherent. 2. Pin point perforation opposite the spine of Henle. 3. Cortex of medium thickness. 4. Cells large. 5. Cavity at the tip and small cavity opposite the spine of Henle. The rest of the cells were soft and filled with exudate but not broken down. No temperature present during the study of this case.

CASE 5: A. S., age 19 years. (12-23-22.)

History: Patient had grip ten days ago and three days later the nasal discharge became thick and profuse. On this day she sniffed some salt solution so as to wash out nose when she felt a click in the right ear and pain ensued.

Examination: 1. The patient looked toxic, sordid lips, dry coated tongue, eyes cloudy, nose stuffed and repeated blowing. 2. Bulging ear drum was incised under ethyl-chlorid anesthesia, and pus was present. 3. Palpation: Pain all over mastoid upon light manipulation. 4. Hearing tests: Weber lateralized to the right; Rinne negative; Schwabach plus.

The discharge continued for 11 days without cessation. The pain became unbearable, hearing almost completely lost. Whispered voice not heard at all and spoken voice only heard within two feet.

Laboratory Findings: Leukocyte count 14,000; Urine negative. At this time the periosteum felt thicker with a loss of the post auricular sulci. Tenderness all over the mastoid.

X-ray Report: Pneumatic type. Large activity extending from the knee of the lateral sinus to the tip and extending back over the lateral sinus. Anterior wall o the sinus cannot be seen.

Operative Findings: 1. Periosteum thickened, adherent and stripped with difficulty. 2. Cortex very thick. 3. Large cavity at the tip with some absorption around the zygomatic area. 4. Cell extension was marked and all cells were filled with granulation tissue, with a small cavity posterior to the lateral sinus. The lateral sinus was exposed and covered with granulation tissue which was not disturbed.

CASE 6: Mr. D. R., age 73 years. (3-19-23.)

History: Patient was a hard working sturdy farmer. Three months prior to examination he gives an indefinite history of having had pain in the right ear. He was seen by a local physician. At no time did he have a discharging ear. He suffered pain for about four weeks before he came to the city to seek advice and relief. He was seen by a general practitioner. Neurologic examination was negative. Urine negative. Blood pressure normal. No evidence of optic atrophy. His chief complaint was pain over the squamous portion of the temporal bone. He was shaved and leeches were applied for two days. He was relieved of pain for several days but the pain returned more severely. It was then I was called in consultation.

Examination: 1. Severe continuous headache over right side, especially over squamous portion of the temporal bone. 2. Patient felt as though someone were pulling constantly at his head. 3. Ear drum negative. 4. Hearing tests: Loss of low fork; Loss of whispered voice; Weber lateralized to the right; Rinne negative; Schwabach increased; MacKenzie plus. There was a slight post-auricular edema caused by the leeches. Inspection therefore was of no value. There was severe pain on deep palpation over the zygomatic and squamous portion of the temporal. Laborative findings negative.

X-ray Report: Mastoid of the pneumatic type extensive in area on the normal side (left) clearly defined. On the right side the mastoid area shades off into the surrounding tissues, as though the surrounding bone had been partially absorbed. The trabeculae can be recognized at places, and at others seemed to be totally lost.

Operation: Local anesthesia. 1% novocain and 1½ ounces used.

Operative Findings: 1. Periosteum thickened and adherent. 2. Cortex very thick. 3. Cells very large and pus under pressure and pulsating. 4. The whole mastoid cavity was a mass of liquified tis-

sue. 5. The sinus was completely exposed and covered with granulation tissue. There was also an extra dural abscess in the middle fossa. I was unable to explain the shadow around the parietal region which I saw on the X-ray plates, as the bone looked healthy and I did not follow that course through; unless the shadow was due to the pus pocket which emptied itself when the cortex was removed. 6. The necrosis extended about ½ inch posterior to the lateral sinus. 7. The mastoid cavity was very deep and wide.

CASE 7: T. S., age 23 years. (3-9-23.)

History: Patient was sick with grip and after four days developed acute suppurative otitis media as a complication. He developed severe pain in the right ear which became progressively worse.

Examination: 1. Nasal congestion and profuse thick nasal discharge. 2. Ear examination: Found three distinct discolored bluish blebs in different parts of the ear drum. These were punctured and fluid trickled out after which the ear drum came into view and was found to be sagging and when incised there was a flow of free pus. The ear drum was reincised at different intervals so as to promote drainage, but there was no abatement of the symptoms. This continued for about 3½ weeks when the sharp lancenating pains were displaced by a hemicrania, a loss of hearing and a heavy boring pain. His general condition became annoying but not alarming. 3. Chest examination revealed fibrosis with some activity although sputum was negative for T. B. 4. Examination on day of operation: Profuse aural discharge which was pulsating. Sagging of posterior superior canal wall. 5. Fork tests: Loss of whispered sound; Loss of low tone fork; Spoken voice audible at 7 feet; Weber lateralized to the right; Rinne negative; Schwabach plus; MacKenzie paradox positive for 4 seconds; Temperature normal. Laboratory tests: Leukocyte count 11,200; Urine analysis negative. Inspection: Loss of post-auricular sulci. No apparent displacement of the auricle. Palpation: Periosteum over mastoid thickened and seemed adherent while on the normal side the skin was readily movable. Superficial palpation. No pain. Deep palpation. Pain over tip and pain at posterior edge of mastoid at the point of the emissary vein.

X-ray Report: Mastoid of the pneumatic type, large cells, showing destruction about knee of the lateral sinus and also extending posteriorly to the lateral sinus.

Operative Findings: 1. Periosteum thick and adherent. 2. Cortex thick. 3. Superficial layer of cells of medium size slightly congested but no liquifaction present. 4. Second layer of cells filled with pus and granulation tissue was found only in certain

areas. There was a tendency to coalescence. 5. Cavity at the tip. 6. Peri-sinus abscess and anterior bony wall of sinus completely gone with a large cavity posterior to the sinus. 7. Sinus covered with a bluish granulation tissue which was not disturbed. 8. Pus found in these cavities was thick but not under pressure.

Conclusion: In this mastoid there seemed to be three strata of cells. The upper strata was almost healthy, the second strata was filled with pus and granulation tissue, while the floor of the mastoid had all the pathology. This mastoid was deep, very wide and the cellular distribution was extensive. Local anesthesia, novocain 1% and 1½ ounces were used.

CASE 8: S. S., age 16 months. (7-9-23.)

History: Patient had acute rhinitis which terminated in acute suppurative otitis media, bilateral. Both ears were incised and free pus found. The discharge and toxic temperature lasted for six weeks.

Laboratory: Leukocyte count 22,000, 28,000, 21,500. Urine analysis: Faint traces of albumen.

X-ray Report: Mastoid poorly developed, though normal for age. Lime absorption in both sides in the region of the antrum, more on the left than on the right.

Examination on day of operation: 1. Profuse aural discharge and pus under tension. 2. Sagging of the posterior superior canal walls. 3. Restlessness and constant crying. 4. Post- auricular tenderness all over mastoid. More marked on the left side, especially over the zygomatic area.

Operative Findings: 1. Bilateral mastoidectomy performed. 2. Periosteum thickened and somewhat difficult to strip. 3. Cortex looked good and of medium thickness. 4. Pus thick and under pressure. 5. Discreet areas of liquifaction. 6. Dehisence of dural plate about the size of a dime. (Left side.) 7. Mastoid cavity very deep, about 1 inch in length and ¾ of an inch wide. 8. Both antra were deep and extensive necrosis was found around the antra.

CASE 9: L. P.. age 15 months. (3-18-22.)

History: This child when first seen, had a temperature of 104°. She was very restless, and looked ill. There was a history of intestinal disturbance with flatulence. One week previous to this date the baby was seen by a peditrician who incised the right ear drum but there was no discharge. Five days later the left ear opened spontaneously. Examination of the ear revealed a sagging in the postero-superior canal wall of the left ear, with profuse discharge. The right ear drum revealed a linear incision with a thin serous dis-

charge. I watched the baby for four days consecutively without abatement of the symptoms. On March 22, 1922, a post-auricular examination disclosed one crease on the left side and 2½ creases on the right side. At this time I found that there was a slight thickening of the periosteum, and immobility of the skin over the left mastoid. I sent the baby into the hospital and ordered both ears X-rayed. The pictures showed the semi-circular canals in situ. The leukocyte count was 25,000. The mastoids were re-photographed three times and at no time could we make a definite diagnosis from the X-ray readings. I did, however, note that on palpation, especially on deep pressure over the left mastoid, the child made an attempt to pull my hands away, and her cries and contortions became more aggravated. On March 25, 1922, although at this time there was some slight tenderness over the right mastoid, I decided to open the left mastoid and found it to be completely broken down. The temperature subsided and on the fourth day it reached normal. On the eighth day, I found both ears completely dry. The baby was discharged from the hospital April 5, 1922. On April 15th, I again was called to see the baby who was being attended by a pediatrician for the past five days, treating the intestinal tract The same history of intestinal disturbance with flatulence was given and all our efforts were concentrated toward the intestinal tract.

Examination of both ears: I found the left ear dry and the mastoid wound clean. The right ear revealed a scar from the previous incision.

Post-auricular Examination: There was no displacement of the auricle but there was a smoothing out of the folds, leaving only ½ of the post-auricular fold intact. I saw no urgency to return the baby to the hospital, as our X-rays were negative. We did, however, find some tenderness over the mastoid, especially on deep palpation over the antrum and at the tip. The leukocyte count at this time was 15,000 and the differential, was 80% polymorpho-neuclars. With a low leukocyte count and high differential cell count and with a history of previous suppuration and sudden cessation, a loss of post-auricular creases, tenderness on deep pressure, and with the previous hazy picture of intestinal disturbance, I felt that we should not lose sight of the fact that the right mastoid might be involved. On April 20, 1922, I ordered another X-ray of the right mastoid. The cellular structures looked blurred but there was no evidence of any breaking down. There was no history of any chills, but the temperature continued to range between 100 and 104°. On the 26th, I again met the pediatrician and we went over the case together and after a long conversation, we decided that the flatulence, although

it persisted was not enough to explain the toxic symptoms that the child presented. We decided to operate on the right mastoid. The leukocyte count was 15,000 and the poly-morphoneuclar was now 80%. A post-auricular incision was made and I found the periosteum thickened and adherent. There was a thick cortical layer which I removed. I found the pus under tension and the mastoid completely broken down. The baby made an uneventful recovery. This patient taught me a lesson that the clinical picture should stand out primarily.

X-ray Report: Mastoid cells undeveloped. No evidence of disease shown. Semi-circular canals clearly shown.

Note: The semi-circular canals were visible in the plate because in children there is very little lime salt and the rays therefore penetrate through everything until they reach the hard bony structure.

Conclusion: The clinical picture is an important factor to be borne in mind, as you will note the X-ray findings do not coincide with the clinical and operative findings.

CASE 10: S. P., age 11 years. (1-23-23.)

History: Patient had rhinitis for several days and after forceful blowing of the nose developed pain in the left ear. He presented himself at the office Jan. 23, 1923 with a profuse aural discharge from left ear drum. During the interval between January 23rd and February 22nd, I only saw him twice. On February 22nd, the discharge became very thick and lessened. On February 26th the discharge practically ceased but the pain over the mastoid increased. Upon examination I found that there was only a small linear perforation in the ear drum with thick pus exuding. There was a loss of the post-auricular sulci and thickening of the periosteum. On February 27th, auricular displacement was noted and the tenderness over the mastoid became more marked. His hearing was impaired and he at no time had any temperature.

Laboratory Findings: Leukocyte 11,200; Urine negative.

X-ray Report: Mastoid on the right side, large, pneumatic in type, with medium size cells. Healthy. Left side shows very much contracted mastoid of the diploeic type with evidence of disease located at the knee of the sinus and extending down to the antrum. The lateral sinus is clearly outlined, while in the right side it can scarcely be recognized.

Operative Findings: 1. Periosteum thickened and adherent. 2. Cortex thin. 3. Fistula present opposite the spine of Henle and cavity underlying it. 4. Pus thick but not under pressure. 5. The

rest of the cells were filled with pus, granulation tissue, and most of the intercellular septa were still present. 6. The mastoid cavity was large, deep and I was unable to locate the antrum so I closed the wound completely.

Laboratory Report: Pus returned sterile, no growth on blood agar and Loefflers blood serum.

CASE 11: W. F.. age 1½ years. (1-12-23.)

History: Six months previous to this ailment had acute suppurative otitis media with spontaneous cure. On January 23rd, when baby was first seen by me, I noted an acute suppurative otitis media with a post-auricular edema. This was secondary to measles. Incision revealed a post-auricular abscess secondary to scalp infection.

X-ray Report: Mastoids poorly developed. The right mastoid shows a few cells. The cell walls are not clearly defined. The left mastoid shows no evidence of cells and is apparently of the disploeic type, or possibly sclerotic. Signs of disease indefinite.

Conclusion: The post-auricular abscess produced a shadow, and the previous suppurative otitis media caused a slight sclerosis, which in this instance could have been misinterpreted for some serious pathology.

CASE 12: E. S., age 9 years. (12-22-22.)

History: This patient had an acute suppurative otitis media, complicated by rhinitis with spontaneous rupture of the ear drum. When first seen, she had profuse discharge and severe pain in the ear.

Examination: Impaired hearing; Loss of whispered sound; Spoken voice audible at 3 feet.

Laboratory Findings: Leukocyte count 9,000; Urine negative.

X-ray Report: Left mastoid well developed, pneumatic in type, large cells. Area extensive. Right side shows mastoid pneumatic in type. Cell walls indistinct and at places entirely destroyed.

Operative Findings: 1. Periosteum thick and adherent. 2. Cortex thick. 3. Complete liquification of the mastoid cells with a peri-sinus abscess. 4. Cavity large, and antrum very deep.

CASE 13: Mr. J. C., age 60 years. (3-18-22.)

History: Patient has been ailing and losing weight for the past three years, suffering with diabetes and boils all over the body. He developed pain in the right ear and he called in his son, who is a physician. It was thought at that time that he had furonculosis of

the external auditory canal. After several days of treatment, he consulted a neighboring otologist who incised the edema in the canal and suppuration followed. His symptoms were not alleviated and after 13 days I was asked to see him.

Examination: Profuse aural discharge. Eczema of the external auditory canal and auricle. The pus was pulsating. The ear drum was almost completely destroyed. His hearing was impaired and the temperature was never above 101°. The most annoying symptoms were severe headache and hemi-crania which was worse at night. He was treated expectantly for three weeks, after which he was sent into the hospital for observation. Pain over mastoid on deep pressure only.

Hearing Tests: Loss of low tones; Weber lateralized to the right; Rinne negative. Bone conduction diminished.

Laboratory Findings: Urine, 4% sugar present; Albumen present; A few hyline casts present.

X-ray Report: Mastoid of the pneumatic type. Small cells. Medium size area on the left. On the right the area corresponds to the left. The cell walls are destroyed. The wall of the sinus is indistinct. There is destruction in the region of the antrum, and increased transparency above this area. The oblique view shows thickening of the cortex.

Operative Findings: 1. Thickened periosteum and very adherent. 2. Thick cortex. 3. Large quantity of pus and pulsating. 4. Peri-sinus abscess. The whole sinus was exposed and covered with granulation tissue. 5. Extra-dural abscess with a complete loss of the dural plate from the antrum all along the roof of the mastoid cavity. 6. Many cells filled with necrotic mucous membrane, posterior to the lateral sinus. When found, the mucous membrane was edematous and of a dark bluish color and very thick.

Operation: Local anesthesia, 1% novocain.

Complications: Infection of the neck.

CASE 14: Mrs. M. L., age 48 years. (12-13-23.)

History: Patient had rhinitis complicated by an acute suppurative otitis media. There was a profuse discharge with severe mastoid tenderness and a slight toxic facial palsy. On December 13, 1921, she went to the hospital for observation. After being there for one week her condition cleared up. This again emphasizes the point that large cells may empty themselves. The patient returned to me May 11, 1922, with an acute exacerbation of these symptoms. The ear drum was again incised and X-rays taken. Operation was

suggested but the patient refused. She now has a chronic suppurative otitis media.

X-ray Report: Mastoid of the pneumatic type, large cells, extensive in area. Clearly distributed anteriorly and posteriorly to the lateral sinus. On the left side the cells are filled with exudate but the cell walls are still visible.

CASE 15: H. B., age 2 years. (3-13-23.)

History: Patient had measles complicated by an acute suppurative otitis media, bilateral. Examination revealed profuse aural discharge. Smoothing of post-auricular sulci. Extreme tenderness over mastoid on palpation. Displacement of right auricle.
Laboratory Findings: Leukocyte 26,000; Urine negative.

X-ray Report: Mastoid cells unusualy well developed for this age. Both sides show disease. On the right the disease is present in the neighborhood of the knee, indicated by a general clouding. On the left the disease extends out into the zygomatic area with destruction of the cell walls.

Operative Findings: 1. Right side: Periosteum thin, stripped very easily. 2. Perforation of cortex on the right side with extensive necrosis over the zygomatic region. 3. Left side: There was liquifaction of the zygomatic cells and there seemed to be a distinct bony shell dividing the zygomatic cells from the rest of the mastoid cells.

CASE 16: B. F., age 5 years. (3-15-23.)

History: Patient had measles complicated by otitis media, bilateral. There was spontaneous rupture of both ear drums. When seen, there was a profuse aural discharge with septic temperature and pain over both mastoids. Both ear drums were incised three times. This condition continued without improvement. There was a progressive loss of hearing and with the profuse discharge, I felt that an operation was imperative.

Laboratory Findings: Leukocyte count 15,900. Urine analysis negative.

X-ray Report: Mastoids on both sides extensive in area. Both sides show disease. Examination made March 15, 1923 showing a general clouding of the cells on both sides. Cell walls still visible. On the left side there is a definite localized absorption just below the knee of the sinus and extending over the sinus. The cell walls show some absorption on the right side near the apex. March 19, 1923; the evidence of disease and lime absorption has increased on both sides at the area previously described.

Operative Findings: 1. Periosteum thickened but not adherent. 2. Large cavity at the tip on the right side with complete breaking down of the zygomatic cells. Left side: Complete breaking down of the zygomatic cells with extensive necrosis of the cells behind the lateral sinus.

Conclusion: The mastoids were X-rayed, March 15, 1923, and March 19, 1923 (4 days apart). The comparative study showed that there was a progressive absorption taking place during this interval. The operation was performed on the 22nd of that month. (A 3 day interval.) Our operative findings here showed more pathology than the X-ray, but I feel that we are not to condemn the X-ray as there was a 3 day interval and so much more destruction took place.

CASE 17: J. S., age 36 years. (10-22-23.)

History: Patient began with an acute rhinitis and after fourteen days developed pain in the right ear. Four days later he came to my office suffering with pain in the ear and pain over the right side of the head. Examination revealed a bulging ear drum which was incised under ethyl-chloride anesthesia. (This was on October 22, 1923.)

Previous History: Patient gives a history of having had scarlet fever during childhood, and also a discharging ear. On November 23, 1923, he returned complaining of severe pain over mastoid. He had a profuse aural discharge and a marked loss of hearing.

X-ray Report: Mastoids of the pneumatic type. Symetrical. On the right side there is evidence of disease, showing at the tip and involving the area posterior to the lateral sinus. There also appears to be disease in the region of the antrum.

Operative Findings: 1. Periosteum thick and adherent. 2. Cortex very thick and sclerosed. 3. After deep chiseling, we found an area of necrosis at Macewen's triangle. 4. Liquifaction of the deep cells in the zygomatic region. 5. Cavity at the tip and a large cell filled with pus posterior to the lateral sinus. 6. Lateral sinus exposed and covered with granulation tissue. Temperature normal.

Conclusion: Here we have a history of inflammation and discharging ears during childhood which probably explains the sclerosis of the mastoid bone. Only the deeper layer of the cells were involved, as there were no superficial cells. Clinically the tender points corresponded to the X-ray and operative findings. These tender points were at the tip, over the posterior edge of the mastoid and over the zygomatic area.

CASE 18: F. C., age 5 years. (5-26-23.)

History: Patient had grip and rhinitis complicated by acute suppurative otitis media. Both ear drums were incised four times without abatement of the symptoms. Child was toxic and very tender over both mastoids. He had profuse aural discharge and pain over tip on the right side and pain over emissary on the left side.

X-ray Report: Mastoids of the small cell pneumatic type. Rather extensive in area, considering the age of this child. A definite clouding of the cells on the right side. Cell walls still visible. Left side shows destruction of the cell walls about the tip.

Operative Findings: (Bilateral operation.) 1. Right side: Periosteum not thickened and not adherent. 2. Cortex medium. Cavity at the tip. 4. The rest of the mastoid cells were soft, filled with pus, but not liquified. 5. Left side: Nothing unusual about periosteum on this side. 6. Cortex congested and of medium thickness. 7. Cavity opposite spine of Henle. 8. Small cavity at the tip. 9. Zygomatic cells necrotic.

Conclusion: In bilateral suppuration, it is natural to operate on the side that shows most symptoms and most destruction on X-ray plates. During this operation, I associated the shadows seen on both plates and since both mastoids are usually alike, I felt that I would find sufficient pathology to warrant mastoidectomy.

CASE 19: I. S., age 72 years. (1-25-24.)

History: Patient was ill with pneumonia for three weeks and suffered with pain in the ear for one week. Ear was incised by another otologist and pus was found.

Examination: 1. Profuse aural discharge. 2. Pain all over mastoid. 3. Thickening of the periosteum over mastoid. 4. Nipple-shaped ear drum.

X-ray Report: Mastoid of the pneumatic type, extensive in area. Symmetrical. General area clouded by exudate. There is absorption of lime salts in the neighborhood of the lateral sinus and extending posteriorly.

Operative Findings: 1. Posterior thick and stripped with difficulty. 2. Cortex thick. 3. Pus under pressure. 4. Mucous membrane very thick, and bleeding from the bone was very profuse. 5. Sinus superficial when broken into. 6. Large cavity posterior to the lateral sinus. 7. Extensive necrosis of the posterior canal wall.

Operation: Local anesthesia, 1% novocain.

CONCLUSION.

1. X-ray is indicated as a routine whether the diagnosis has been made or not, so as to correlate clinical and operative findings.

2. It is important to cleanse the area to be X-rayed as ointments used as medicaments may contain some metallic substances which will produce pseudo shadows.

3. Some atypical cases probably would never be diagnosed without an X-ray where we had no middle ear suppuration and at operation we found an extra-dural abscess.

4. X-ray in my experience has always been very helpful and at no time was I inclined to operate unless the clinical symptoms warranted such procedure.

5. The mastoid pathology can be watched as to progress by means of X-ray at three day intervals.

6. Those of us following the conservative treatment in mastoid therapy will bear in mind that it is important to keep vigilant watch on the lateral sinus. Should there be any signs of haziness or complete loss of the lateral sinus from view, we may suspect necrosis of the anterior bony wall with a peri-sinus abscess.

7. It is especially important for the novice beginning to practice in our specialty to take routine X-rays as he may learn the topographical and regional anatomy of the mastoid, as to type, whether cellular or non-cellular and position of the lateral sinus.

8. The angle from which the mastoid picture is taken must be understood as otherwise the clinician will misinterpret some of the shadows.

9. Cavity formation appears as a blurred area without septa and the edges of the cavity are not distinctly visible. This must be differentiated from a large normal cell which is irregular in shape, the edges of which are distinct, sharply outlined with septa intervening.

10. In order for Dr. Pfahler to obtain good pictures in children, I ask him to administer a few whiffs of chloroform.

11. In children where cells are not fully developed, I always study the plates along the posterior canal wall, as in this area the bone is thickened and the antrum has one or two cells and if there is any disease it should be seen there. This condition was especially true in case No. 9. In this case the semi-circular canals were distinctly visible and the only pathology noted was around the antra.

12. In mastoid disease always X-ray both mastoids and compare the normal side with the diseased. In most instances the cellular distribution and general confirmation of the mastoids are alike and any deviation from the normal will readily be seen unless there is a history of previous middle ear suppuration.

13. In bilateral suppuration both plates should be studied, selecting the side to operate which shows most pathology from a clinical and radiographic standpoint.

14. The operative findings in one mastoid will usually interpret the plates of the oppoite side and a decision as to the course of pursuit will be simpler.

15. It is the idea of many clinicians to X-ray all mastoids so as to correlate the X-ray with the clinical and operative findings. Although we may make our diagnosis in most cases clinically and judge whether operation is indicated or not, it should however be our aim to develop clinically so as to be able to outline the pathological conditions underlying the mastoid cortex.

16. Repeated X-rays will show whether the disease is at a standstill, progressing or undergoing sclerosis.

17. We must bear in mind that operations are often delayed after X-ray plates have been taken and radiographic studies must therefore not be condemned because they do not coincide with the operative findings.

I desire to cordially express my sense of indebtedness and gratitude to my friend and able chief, Dr. George M. Coates, to Dr. Benjamin Shuter for his valuable assistance, and Dr. George P. Pfahler for his able and conscientious aid and collaboration.

1915 Spruce Street.

STUDIES OF MASTOID DISEASE BY THE X-RAYS WITH OPERATIVE FINDINGS. DEMONSTRATION OF A SPECIAL LOCALIZER.*

DR. G. E. PFAHLER, Philadelphia, Pa.

In most large otologic clinics the examination of the mastoid by means of the X-rays has become almost a routine procedure. The value placed upon these studies in each clinic is generally measured by the technical skill which has been developed in making Roentgenograms of good quality, but more particularly by the intelligent and skillful interpretation. The clinicians who have had the most experience with this aid in diagnosis are the ones who are most enthusiastic. I know of no Roentgenologist who will ask a clinician to depend entirely upon the Roentgenographic evidence in mastoid disease, especially in deciding upon operation.

The X-ray picture of the mastoid is often interpreted with difficulty, and requires keen observation, a thorough knowledge of the conditions which cause variations in density and a consideration of the technique with which the film was made. There is almost as much variation in the type and distribution of the mastoid cells as there are variations in the type of noses on people's faces. For this reason[1] both mastoids are always studied. Generally they are symmetrical, but Dixon in a study of 522 cases found asymmetrical mastoids in 14.5%. Auberg[2] in a study of 120 pairs of mastoids found 82 symmetrical and 38 asymmetrical.

Law[3] divides the mastoids for Roentgenographic studies into the "Non-cellular" and the "Pneumatic or cellular". The "Non-cellular" consist of three types, the undeveloped or infantile, the diploetic and the sclerotic. The pneumatic or cellular consist of the large cell, the small cell and the mixed cell. These two general types may be mixed, and rarely one may have a penumatic type on one side and a diploetic type on the opposite side. Cheatle[4] calls especial attention to the infantile type which existed in 20% of all the cases he examined and persisted into adult life in one or two per cent. The common characteristic of these infantile types is the dense outer antral wall. The lateral sinus is generally much farther forward than in the cellular types. Gerber[5] cautions against interpreting

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these infantile types as "sclerotic" and says that in the infantile type, a chronic infection can be prevented by early drainage of the antrum, regardless of the absence of the classical mastoid signs.

This variation makes interpretation difficult. Spencer[6], in a review of the X-ray reports of 100 cases of mastoiditis, as Base Hospital, Camp Lewis, Washington, found the X-ray reports confirmed at operation in 80%. The 20% included all doubtful cases and all cases in which the X-ray report or the operative report was very briefly or incompletely written. He believes that if both reports

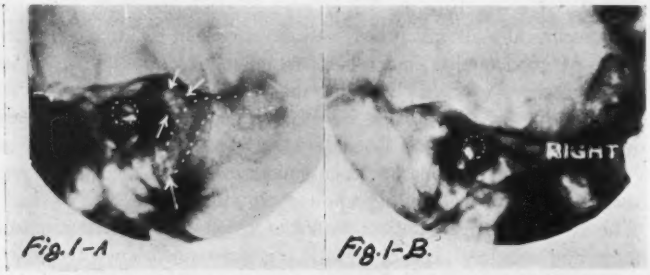

Fig. 1. (Case 2) Age 6. Mastoid of pneumatic type, more fully developed than usual at this age. Left side shows lime absorption about the knee at the tip. Lateral sinus clearly outlined. Outline of external auditory canal and the lateral sinus is dotted. A.—Left. B.—Right.

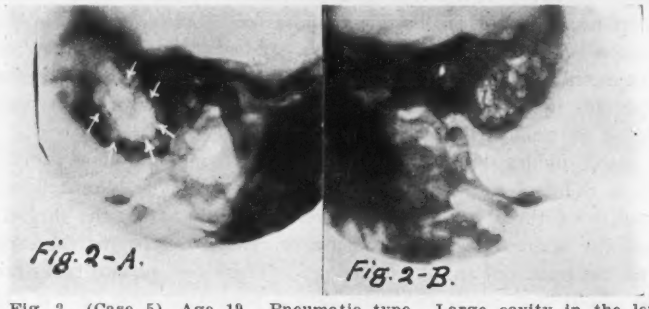

Fig. 2. (Case 5) Age 19. Pneumatic type. Large cavity in the left extending from the knee of the lateral sinus to the tip and extending back over the lateral sinus. Anterior wall of the sinus cannot be seen. A.—Right, diseased. B.—Left, normal.

had been completely written there would have been confirmation in a much larger percentage.

In the series of cases studied by Dr. Ersner and myself, the rays failed to show disease in a child of 16 months and in a man of 73 years. In the case of the old man the disease was not recognized in the X-ray film but a review of the films shows the evidence of the disease as was found at operation. In the case of the young child

the failure to show disease was due to lack of development of the mastoid cells. In the case of the child even the semi-circular canals were shown, yet no evidence of disease could be recognized. Law says, "Except in very unusual cases the mastoid process does not show cellular development before three years of age, and usually not until about the fifth year of age". We are glad to say that in a number of cases of children, we have been able to recognize disease under 3 years of age.

A satisfactory Roentgenogram should show the external auditory meatus, the internal meatus, the mastoid antrum, the temporo-maxillary joint, usually the outline of the lateral sinus, as well as the middle and posterior cranial fossa. Frequently, the ossicles of the ear are shown and at times in children the semi-circular canals. One should always keep in mind the type of mastoid, and this should be described definitely.

TECHNIQUE.

The first essential in the Roentgenographic examination of the mastoid is the making of good films, which will show the finest possible details. The second essential is skillful interpretation, and the third essential is the correlation of the X-ray evidences with the clinical symptoms. Insofar as any of these three are lacking, there will be failures.

1. The patient's head must be absolutely fixed. We accomplish this with sandbags.

2. The central ray should be passed directly through the auditory canal. This I have been able to accomplish by means of a special localizer which I am demonstrating to you and which will be described in detail in a subsequent paper. Briefly, it consists of a head rest which permits us to absolutely fix the external and auditory meatus in direct relation to the tube holder, so that the two sides of the head can be examined exactly the same and any two heads can be examined alike. The central ray is passed obliquely from above at an angle of 10 degrees, and posteriorly at an angle of 15 degrees. This makes the central ray enter the proximal side of the head about 1½ inches above the external auditory meatus about 1¾ inches posteriorly to the external auditory meatus. The films may be made stereoscopically, as is recommended by Allen[7] and others. We do not make stereroentgenograms routinely because this of necessity changes the direction of the central ray. In certain special cases stereroentgenograms are of advantage.

3. Sharpness in detail is improved by the use of a small cylinder diaphragm. We use one cylinder 3 inches in diameter at the sur-

face of the head and then make a second film on each side with a cylinder 1¾ inches in diameter. Such small diaphragm work is only possible where an exact localizer is used.

4. The duration of the exposure will vary with the individual technique. We use super-speed films, double screens, a 3½ or 4 inch parallel spark gap, and 4 to 6 seconds exposure.

In addition to the positions described above, one may often get special information with regard to the tip of the mastoid by direct-

Fig. 3. (Case 14) Age 48. Mastoid of the pneumatic type, large cells, extensive in area. Clearly distributed anteriorly and posteriorly to the lateral sinus. On the left side the cells are filled with exudate but the cell walls are still visible. Examination December 13, 1921, shows early stage of mastoid disease.

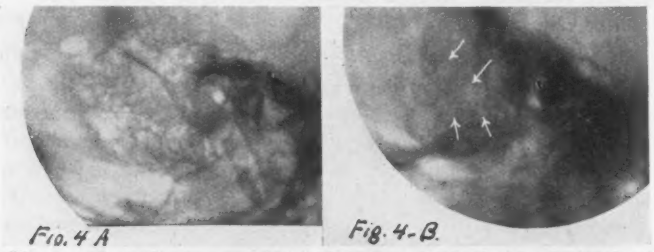

Fig. 4. (Case 19) Age 70. Mastoid of the pneumatic type, extensive in area. Symmetrical. General area clouded by exudate. There is absorption of lime salts in the neighborhood of the lateral sinus and extending posteriorly. A.—Left, normal. B.—Right, diseased.

ing the central ray through the mouth toward the tip on the opposite side. In whatever position, both mastoids should be examined and compared.

Hickey[8] has described an antero-posterior position for the mastoid. Each side is exposed separately with the patient lying on his back. The tube is tilted 5 degrees. The central ray is passed through the tip and a short exposure is made.

Sometimes additional information may be obtained by oblique exposures, either projecting the shadow of the skull forward, by directing the central ray from behind and from below, or one may

project the cells into the accipital region by directing the central ray from above and anteriorly to the petrous portion of the temporal bone on the proximal side. This position will project the dense petrous portion below the level of the antrum.

Gerber has described, but not yet published what he calls the "Occipital Projection", which is obtained by introducing the central ray nearer to the occiput, and obtaining more detail about the antrum.

INTERPRETATION.

It is usually necessary that the clinician correlate the clinical symptoms with the interpretation by the Roentgenologist. However, it is not always necessary that the clinical symptoms correspond to the Roentgen ray findings. In some cases the local clinical symptoms may be absent. Lange says, "The mastoid may be the seat of an infection of either recent or remote origin, unheralded by a single symptom", and says that no less an authority than Politzer made the statement that the most extensive necrosis may occur in the mastoid without giving rise to a single symptom.

Since about 85% of the mastoids are symmetrical, one can generally make a direct detailed comparison of the two sides. When both sides are diseased, or when there is symmetry, the Roentgenologist must use his general knowledge of diseased appearances as compared with the normal in the same mastoid, as well as in other patients. The normal mastoid of whatever type gives the appearance of a network of continuous cancellus lines, corresponding in density to the surrounding healthy bone. The general evidence of disease is indicated by exudate in the cells, by destruction of the cell walls, or by evidence of repair as is shown by sclerosis. It is necessary for the surgeon to know the type of mastoid, the degree of involvement and the presence or absence of bone destruction.

Acute Mastoiditis: In the minor grade, there is a cloudiness of the cells, with the cell walls or trabeculae still clearly visible. One must be quite sure that the cloudiness is not due to a faulty or fogged film. The cloudiness may also be due to swelling in the soft tissues as from furnuculosis. The sinus groove stands out more clearly, and in early acute mastoiditis, the region of the antrum is relatively more opaque than the body of the antrum. It must be borne in mind that this cloudiness may be due to pus or to thickened mucous membrane or granulations.

Severe Acute Purulent Mastoiditis: The pneumatic type show a marked degree of opacity over the entire mastoid area. The cell walls may be completely obliterated, and one detects evidence of

absorption of lime salts. This indicates severe active disease but not always necrosis or destruction of the cell walls. If the absorption of lime salts is localized, or if the cell walls are totally destroyed it is likely to indicate necrosis. The disease is most likely to be shown in the antrum or at the knee of the sinus. In the stage of lime absorption, the anterior wall of the lateral sinus is apt to become less distinct, because of absorptions of lime salts.

Mastoid Abscess: Abscess is indicated by a destruction of the cell walls, which may involve the entire mastoid. If it occurs about the wall of the lateral sinus it becomes a peri-sinus abscess. It may break through the cortex and give local external sypmtoms, or it may break through the tegmen tympani and become an epidural abscess. The abscess is indicated by an area of increased transparency as compared with the surrounding tissues.

Chronic Mastoiditis: This condition is indicated by a general sclerosis, very similar to an undeveloped mastoid. A close search should be made for any remaining cells, or any evidence of bone necrosis or sequestrum. The history of a persistent or chronic recurring purulent discharge will serve to differentiate this condition from the undeveloped mastoid.

Cholesteatoma: The process is inflammatory and the edge of the cavity show an osteitic change, a thickened border indicated by increased density. Law says "Cholesteatoma always occurs in a chronic process or in an undeveloped or sclerotic type of mastoid." The discharge is usually foul and will show the presence of cholesterin crystals.

In this clinical study of 142 cases in association with Dr. Ersner, many of the films were made at the Medico-Chirurgical Hospital and interpreted by my associate, Dr. B. P. Widman. In only two instances did the operator fail to confirm the X-ray findings. In one of these, the child was only 3 years of age and the mastoid cells were undeveloped. In the other case the disease was shown in the films and can be seen now, but was not properly interpreted. This simply indicates that skill is required in interpretation, and is no reflection on the method. In all instances the disease was suspected by Dr. Ersner from the clinical symptoms. This means a confirmation in about 98%.

In a series of cases studied at the Poly-clinical Hospital where most of the films were made and interpreted by my assistant, Dr. William Campbell, with review by myself, Dr. Gleason tells me that in practically all cases the diagnosis was confirmed at operation.

CONCLUSIONS.

1. The X-ray examination should be a part of the routine study of each case of mastoid disease.

2. In all instances the evidence should be correlated with the clinical evidence of disease, especially in deciding upon operation.

3. Success depends first upon skillful technique in making fine Roentgenograms, and secondly, in careful observation and interpretation.

4. The two sides should always be examined, and generally compared but they are not always symmetrical and interpretation must often be made independently of the opposite side.

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BRAIN ABSCESS OF PARANASAL SINUS ORIGIN. TWO CASES.*

DR. GORDON BERRY, Worcester, Mass.

The following two cases of brain abscess of paranasal sinus origin present unusual features which were not entirely cleared until autopsy. The first case had a chronic tempero-sphenoidal abscess of sphenoidal origin but giving no trouble until two weeks before his admission to the hospital in a semi-conscious condition. Operative interference failed to properly drain the abscess and death ensued six weeks later. The second case had an acute frontal lobe abscess of ethmoidal origin. Operative interference succeeded, the patient lived four years and died of a recurrence. With this post-mortem evidence let us review the outstanding features of each case in their chronological development.

Case 1. (F. C.) A strong, vigorous white male, 44 years of age, was admitted to the Worcester Memorial Hospital on November 1, 1922, in a semi-conscious condition. Healthy until two weeks previous, he then began to have severe headaches accompanied by a blurred and double vision. He grew rapidly worse. During the first three days of his stay in the hospital he had several spastic convulsions accompanied by jaw fixation and unconsciousness. The history told of a "sunstroke" seven years previously, and of a gradual failing of the left hearing a little later while working in an army boiler factory. He gave a history of no eye trouble, no ear or nose infection, but an occasional sore throat and once or twice a "quinzy" sore throat with the escape of foul pus. Now, his severe parietal headaches, more marked on the left, a choked disc of four diopters and retinal hemorrhages on the left, a blurring of vision, a greater spasticity of the right side during the convulsions, all pointed to some tumor process of the left base or left tempero-sphenoidal lobe. The pulse was bounding, the temperature around 99°. A decompression operation was decided upon when the nurse noticed a coughing up of some thick, foul pus, with an almost immediate relief of his brain pressure symptoms. At this time the writer first saw the case. In addition to the above localizing signs, he noted a tactile partial anesthesia of the left nasal mucous membrane and of the left cheek (nerve V, divisions 1 and 2). The tongue pointed a

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little to the right (nerve XII). The left cochlea and labyrinth were dead, showing no response to stimuli while the right side was normal (nerve VIII). There was outward past pointing to the left. The Wassermann test was negative and the left acoustic paralysis was provisionally ascribed to his work in a boiler shop. There was some congestion of the fundus and some visual blurring on the right (nerve I). The diagnosis was thus made that we had an abscess in the middle fossa, near the optic chiasm, on the left, probably near the pituitary, and periodically draining into the sphenoid with consequent cranial pressure relief. There was however no especial clouding of the sinuses in the X-ray films and locally there was only a little mucoid discharge from both upper nares, suggesting a slight acute "cold". If anything the left sinuses were a little more involved, both clinically and in the picture. There was no sinus pain or tenderness.

Operation. Under local anesthesia the left middle turbinate was removed and the posterior ethmoidal cells and the sphenoidal sinus freely entered and exposed. No diseased condition of the bone, no granulation tissue, no pus was noted. That portion of the posterior wall of the sphenoid and ethmoid, represented by the anterior face of the sella turcia, and the roof of the sphenoid and ethmoids, was clearly seen and probed. The bone was solid, the membrane appeared healthy. Then part of the right middle turbinate was removed in order to permit free inspection of the right posterior naris. Neither now nor later did more than a congestive mucoid secretion show on this side. The right ethmoidal nerves were much more sensitive than those on the left and working on the right put him into a spastic convulsion.

The next week showed improvement. He continued to cough up thick foul pus about every morning. His mind was usually lucid and fairly active. The vision and fundi became better—almost normal on the right. The post-operative nasal congestion made examination of the sphenoid unsatisfactory and I was never able to clearly demonstrate the source of this expectorated pus until autopsy. It came at rare intervals and all at once. On the eighth day when he began to show more brain pressure symptoms, I did a low left sided decompression operation and Dr. B. T. Burley (the neurologist) explored for a brain abscess. Repeated tappings into the frontal and tempero-sphenoidal lobe failed to reach the pus. Twice we obtained the clear fluid of the lateral ventricle. We could not go deeper than this for to reach an abscess through a lateral ventricle is to invite a prompt general meningitis. Two days later we inject-

ed 30 cc. of air into the left lateral ventricle bringing on a temporary coma. An X-ray of the head was immediately taken and the air distension found almost all in the right lateral ventricle. The left ventricle was collapsed because of the near-by abscess. The periodic coughing up of pus continued; the patient seemed to rally. It was hoped that the brain abscess would work to the surface. Then he became more delirous and succeeded in getting his finger under his bandage. The wound became slightly pussy and the brain herniated a little. This discouraged the second tapping of the brain we were

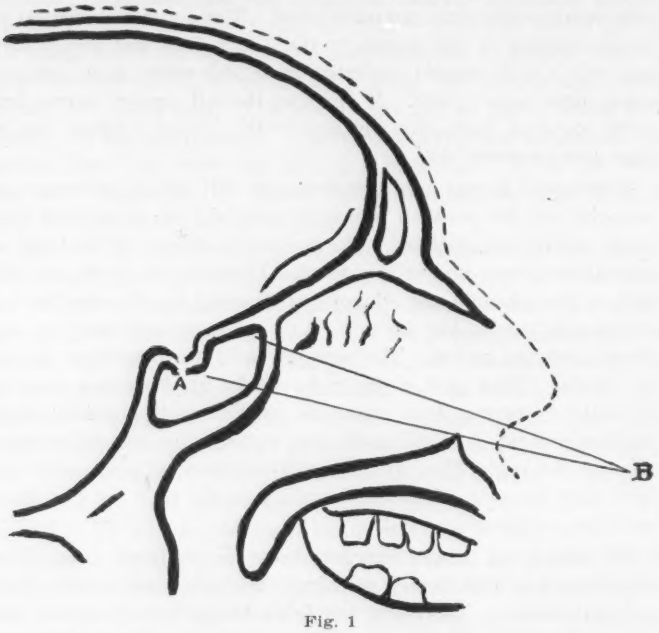

Fig. 1

contemplating. From then on there was a slow, steady decline. The temperature with one day's exception remained between 99° and 100° for five and a half weeks after the operation, then rose in a typical meningitic curve, with death seven weeks after admission.

Autopsy showed a large abscess of the left tempero-sphenoidal lobe below the lateral ventricle into which it had ruptured. It was just ready to evacuate itself at our decompression site when meningitis and death intervened. Its lateral extent posteriorly accounts for his acoustic and labyrinthine paralysis far better than his boiler maker's occupation seven years previously, and we may even question the "sun stroke" at about the same time. An anterior internal

lobulation of the abscess reached to the pituitary fossa, and in the floor of this was found a necrotic opening directly into the left sphenoidal sinus.

Comment. If the writer's reasoning is correct, the process started in the sphenoid. How else can we account for the dehiscence in its roof? Then the sphenoid cleared and remained so; for it looked normal at the time of operation. At that time I failed to find the break in the roof which brings out so important an anatomical point that it will be discussed separately at the end.

Case 2. (Miss G. V.) A well developed Armenian woman of 22, was admitted to the hospital on December 24, 1915, in a partial stupor and mentally unresponsive, with a history of acute left ethmoidal and frontal sinusitis five weeks previously, and a temperature of 102-103°. The temperature had returned to normal, the pain had gone and the profuse nasal discharge was clearing when a severe, almost fatal, epileptiform convulsion came on ten days previous to admission, followed by headache nausea, intermittent vomiting, a slow pulse (66), no fever and an increasing mental indifference to her surroundings. There was a left sided exophthalmos. a suggestion of a left optic neuritis. and a paralysis of the left external rectus.

Her personal history gives a turbinotomy for nasal obstruction five years previously and an early adeno-tonsillectomy. She had been otherwise healthy. Her father died of a shock, the mother had had convulsive attacks, two brothers had had psycho-neuroses for which one had been in an insane hospital. All the children were ambitious, educated and musically talented. There was absolutely no history of any specific involvement and repeated Wassermann tests of the blood and spinal fluid proved negative. We could find no trace of tuberculosis.

On the first day a bony sequestrum, apparently the posterior half of the middle turbinate, was removed from the left ethmoidal region. A little pus was escaping and the patient's condition bettered with this improved drainage. Three days later by the intra-nasal route, the left ethmoidal labyrinth was thoroughly exenterated and the frontal and sphenoid sinuses freely entered. She began at once to improve. The ethmoidal roof was not disturbed until three weeks later when an attempt was made to punch away some granulation tissue which was crowding down from above. This proved to be a brain abscess projecting through an opening in the posterior ethmoidal roof, and the nasal punch removed the abscess floor with a consequent gush of at least two drams of thick pus, followed by a hemorrhage which required temporary packing of the upper naris.

An X-ray at this time showed the abscess cavity filled with air: a large oval area with clearly defined edges, placed vertically in the middle of the left frontal lobe. (See Fig. 2.) She had improved much during these three weeks and now with minor temporary setbacks and no more convulsions she continued to improve until March 17, 1916, after a hospital stay of almost four months, when

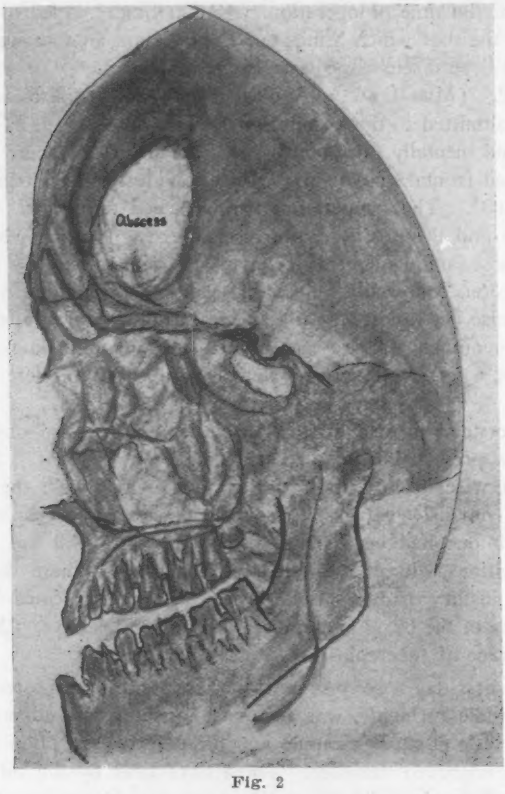

Fig. 2

it was deemed safe to send her home. She had been up and around daily; the eyes had returned to normal; the left nasal discharge had almost ceased; there were no convulsions; she seemed normal.

It later proved that menstruation commenced the morning of her discharge. She felt so well that she started home on the trolley and stopped on the way to visit some friends. On reaching home she found more friends to greet her. She retired at 10:00 p. m. At midnight her mother found her in a convulsion. Five more occurred

during the next three hours. She vomited several times and was semi-conscious the next day. That evening the convulsions came in rapid succession until she was readmitted to the hospital and 35 cc. of negative spinal fluid removed by lumbar puncture; when she vomited, then fell into a quiet sleep. On waking the mind seemed clear but she could not say the proper word—for instance, she called every one by her nurse's name. In spite of morphin subcutaneously and chloral and bromides by rectum she had 35 more convulsions the next night, relieved by the withdrawal of 30 cc. more of negative fluid under pressure. For the first time the white count was now excessively high (31,500) and the urine showed albumen and casts. The convulsions ceased. The next ten days found her out of bed and her mind nearly normal though her memory of what happened during the acute attack was very vague. She left for home in two and a half weeks (on April 17, 1916). She returned to the hospital a little over a month later (on May 28) when she had another series of convulsions lasting twelve hours and clearing under large doses of bromides and morphin, permitting her discharge on June 16. The following month there was a series of twelve convulsions. In November, 1916, she was so well that I felt justified in removing a diseased tonsil stump and some adenoid tissue. In January, 1917, she returned to the hospital where she had about thirty-five more seizures.

Each convulsion began with a slight cry, the mouth opened wide and with a contorted facial expression, the eyes rolled upwards, then the mouth closed. clonic convulsions of the extremities set in, with a laryngeal spasm and consequent extreme cyanosis. The attacks lasted from one to five minutes and were at times so severe as to suggest their proving promptly fatal. A little ether would occasionally stop the attack but usually the ether had to be pushed to an extreme narcosis before it would stop the rapidly succeeding convulsions. After an attack she went into a relaxed comatose state from which she gradually wakened if another attack did not supervene. Scopolomine given in heavy doses was more effective than morphin. At first the attack was followed by mental aberration; later there developed a tendency to a rebellious obstinacy which was quite contrary to her natural sweet disposition.

Then for three years she was free of these seizures. At times there would be a dull headache with slight mental aberrations which her physician, Dr. R. J. Ward, was able to clear by prompt catharsis, rest in bed, and a controlled diet. Thyroid and pituitary extracts were tried. She filled acceptably a responsible position as stenographer and bookkeeper during the day, and studied with her

piano or gave music lessons in the evening. It was hoped that we had won through. Then, after a little initial deafness (the first ear complication experienced) she had a violent left frontal headache and vomited bile on February 2, 1920. Two days later the neck was stiff and the bowels distended with gas. The temperature rose to 104°. There was no convulsive seizure this time. The meningitis was well advanced when she entered the hospital on February 9 and death followed on February 18, 1920, sixteen days after the onset.

The post-mortem examination showed a large oval abscess in the left frontal lobe similar to that we found four years previously, with a generalized meningitis resulting from its rupture at the base.

Final Comment. The abscess in the first case was correctly diagnosed but we could not reach it. The abscess in the second case was suspected but the strong neurotic element with insanity in the family made us think of possible hysterical or epileptiform seizures with the suppurative ethmoiditis as an irritant. Here a kindly chance helped and we successfully drained the frontal brain abscess. But the convulsions did not cease for a year. Some were so severe as to almost kill her. Operative entrance through the frontal table of bone and exploration for a re-formed frontal abscess was repeatedly considered. But it was pointed out in consultation that the former abscess might have left contracting scar tissue which might account for the epileptiform seizures. Operative interference was therefore not undertaken. After over one hundred of these convulsions she apparently got well and remained so for three years of a busy, happy life. Then came the rupture of the dormant brain abscess and death from a rapidly fulminating meningitis.

Anatomically it is interesting to note that the two abscesses went through two of the thinnest points in the roof of the nose. The floor of the pituitary fossa is often thinner than the anterior face in the large type sphenoidal sinus, while the roof of the anterior part of this sinus and of the extreme posterior ethmoidal cells is covered by the thicker bone of the lesser wing. In front of this begins the widest part of the cribriform plate and the neighboring thin bone of the horizontal plate of the frontal, where the second case broke through. With lues and tuberculosis apparently ruled out in both of these cases, it makes an interesting conjecture as to the nature of the infection which caused the bony necrosis and the suppurative extension into the brain. In each case staphylococcus aureus was recovered from the culture.

The first case offers two interesting anatomical points, a more intimate realization of which might possibly have helped. They were

forcibly brought out by my failure to reach the abscess. First, an abscess of sphenoidal origin involving the tempero-sphenoidal lobe must necessarily be internal to the lateral ventricle anteriorly. To tap this abscess, we had three chances. One was through the ventricle which would have drained the pus into the ventricle and caused meningitis at once. We tried the straight approach and stopped when we reached the ventricle. We tried going above the ventricle but the abscess was not so high. We tried going below the ventricle anteriorly but this carried us below the abscess. In theory, a curved trocar passed under the ventricle would have reached it, our second chance. This abscess six weeks later was found at autopsy to extend from the pituitary fossa backwards and outwards under the ventricle so that posteriorly it was situated in the tempero-sphenoidal lobe external to the ventricle. If it was in this position when we tried to tap it, a straight trocar passed backwards near the floor of the tempero-sphenoidal lobe would have succeeded. This was a blind third chance true, but the writer will bear it very carefully in mind if he ever has a similar case.

The second anatomical lesson is learned from the writer's failure to find any break in the roof of the sphenoid until autopsy. During the intranasal sphenoid operation he took away so much of the anterior sphenoidal sinus wall that the neurologist looking over the operator's shoulder himself could see clearly the posterior sphenoidal wall. The membrane was clear and normal. The bone was solid. There was no pus, no granulation tissue seen. He naturally decided against going through this normal bone in his search for an abscess. A study of the lateral X-ray picture of this patient's sphenoid shows a deep pituitary fossa and a sinus which extends not only below this fossa but far back into the body of the sphenoid bone. It was the floor of this fossa which was found necrosed through an autopsy. If we draw a straight line from the nasal vestibule through an imaginary large operative opening in the anterior sphenoidal sinus wall, this line will indicate the visual operative field. We now discover that the opening in the floor of the pituitary fossa was below the operator's line of vision. Only with an S-shaped probe could the writer have reached around this double corner.

Especially when operating by the intranasal route as in this case, much of a large sphenoidal sinus is below the line of vision. In this extreme case, that portion of the roof which was beneath the pituitary fossa could not be seen. A removal of the antero-inferior wall of the sinus might have helped a little.

19 Elm Street.

PROLONGED HEMORRHAGE WITH NORMAL COAGULATION AND BLEEDING TIME!*

DR. JAMES G. CALLISON, New York.

N. F., white, male, age 19, was admitted to the service of Dr. Lefferts McClelland, at the Broad Street Hospital, on August 22, 1923, for an adenoid and tonsil operation.

Personal history (furnished by his mother after operation)—The patient had the usual diseases of childhood—measles, scarlet fever and chicken pox. There was also a history of an umbilical hemorrhage at birth, and a second exsanguinating hemorrhage when he was circumcised at the age of three months. He is said to have had severe hemorrhages when teeth were extracted. At about the age of ten years he had an attack of "rheumatism".

Family history negative (the mother's family all dead and nothing obtainable from her).

Physical examination negative before and after the operation; urine examination negative; Wassermann reaction negative.

August 23, at 9:30 a. m., when he was on the operating table and all was in readiness to start the anesthetic, the patient informed me that he was a bleeder, or had been when he was a child. I delayed the operation long enough to have the laboratory test his coagulation and bleeding time, which were eight and six minutes respectively. The Biffi-Brooks method was used to test the coagulation time. As these results were well within normal limits I proceeded with the operation, believing the story to be one of the usual alarming statements of patients, for which there is no justification. During the operation there was free bleeding which was unusually difficult to control, and he was given a quarter of a grain of morphin subcutaneously. As he had had a sixth of a grain of morphin before coming to the operating table, this made five-twelfths of morphin sulphate as a total. The bleeding was finally controlled, except for a very slight oozing, and the patient was returned to bed. The pulse, taken in the ward, was 108 and fairly good; respiration 12.

At about noon the house surgeon, Dr. Swartz, telephoned that the patient was bleeding freely, and I ordered him to give 20 cubic centimeters of a 30 per cent solution of sodium citrate intraven-

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ously. Neuhof and Hirshfield[1] announce in a recent paper that this is contra-indicated in hemophilia and purpura hemorrhagica, but at this time I had no reason to suppose that I was dealing with either condition. At 2:30 p. m., on my return to the hospital, the patient was cyanotic, pupils pinpoint, respirations of a spasmodic type and about 12 per minute or less. He was bleeding freely from the right tonsillar fossa. This was easily controlled by pressure, but recurred as soon as the pressure was removed. After persisting in the attempt to stop the hemorrhage for more than an hour, two sponges were placed in the right tonsillar fossa and the pillars brought together over them by means of interrupted sutures. This only partially controlled the hemorrhage, oozing continuing. Black coffee per rectum ameliorated the condition of morphin intoxication.

August 24. Slight oozing continued during the night, which became profuse toward noon. All efforts to control the bleeding failing sodium citrate was again injected intravenously, but without effect. As the pulse was becoming poor in quality and very rapid a hypodermoclysis of 500 cubic centimeters of physiological slaine was given and a Murphy drip ordered. This latter could not be given, however, as quite fresh blood blocked the rectal tube and prevented a flow. This within twenty-four hours in a patient suffering from intense morphin intoxication would seem to justify the opinion that there was bleeding from the intestinal tract, in addition to that from the tonsillar fossa, although the fact cannot be proven. At 4 p. m. 20 cubic centimeters of horse serum was given subcutaneously, and by 5:30 the bleeding was reported fairly well under control. On account of the patient's poor condition and because of the fear of a recurrence, transfusion was ordered. This was given at 8:30, the patient receiving 500 cubic centimeters of blood by the citrate method. The patient was a type III and it was necessary to accept a type IV donor for the emergency. A chill lasting twelve minutes followed the transfusion.

August 25. No bleeding. The sponges removed from the right fossa, together with a large clot at the lower pole. The odor from the mass removed was foul and penetrating, and I ordered it thrown away at once, and so overlooked one of the sponges. Following this removal the bleeding was controlled by pressure with dry sponges without difficulty.

August 26. No active bleeding, but a large clot had reformed at the lower pole of the right tonsillar fossa.

August 27. Free bleeding began late at night from the lower pole of the right tonsillar fossa and continued during the night and

the next day. This was not discovered till late morning rounds, as the patient kept swallowing the blood.

August 28. A test taken in the afternoon showed that the patient's hemoglobin was down to 34. Marked tympanites was present, and an enema brought away almost pure blood, indicating the possibility of a second hemorrhage from the intestinal tract, in view of the several days of partial freedom from bleeding from the tonsillar fossa. At 8:30 p. m. a second transfusion was given of 500 cubic centimeters of citrated blood, again from a type IV donor.

August 29. In the afternoon a large clot was removed from the right tonsillar fossa, together with the sponge which had been overlooked on the 25th. After removal of the clot and sponge the bleeding was controlled without difficulty by pressure with dry sponges. Syrup of lactophosphate of calcium was ordered, two teaspoonsfull every four hours, this being less liable to disturb the throat than tablets of lactate of calcium. At 9:30 p. m., profuse bleeding recurred, the patient spitting out large clots of blood in rapid succession. The bleeding point at this time was not determined. The patient was becoming so weak and apathetic that much of his medication was refused, and the appetite was capricious.

August 30. During the day there was no bleeding, but at 10 p. m., the patient again spit out numbers of large clots, the bleeding coming from the left. This continued during the night, in spite of all efforts to stop it. Horse serum was again given, this time intravenously.

August 31. A test taken in the forenoon showed the hemoglobin down to 32. Free bleeding continuing, the third transfusion of 500 cubic centimeters of citrated blood was given at 11:45 a. m., also from a type IV donor. A chill lasting 20 minutes followed. This transfusion brought the hemoglobin up to 45, tested at 2 p. m. A small clot was removed from the border of the soft palate on the left side, at the junction of the anterior and posterior pillars. Bleeding was easily controlled by pressure, and when dry the spot was touched with 50 per cent nitrate of silver. Bleeding again occurred during the night and early morning of September 1st, the patient spitting up large clots of blood.

September 1. When the patient was seen this forenoon, the bleeding had been so profuse during the night that the condition seemed hopeless. Preparations were ordered for the fourth transfusion as soon as the hemoglobin fell below 35 per cent. Dr. Francis Huber advised free administration of fruit juices, believing there might be a scorbutic taint. This was given. Dr. Lionel Rosenberg suggested the administration of 20 drops of fluid extract

of hydrastis together with five drops of adrenalin every four hours, as an old and tried remedy in uncontrolled hemorrhage. This had been ordered two days previously, but owing to a mistake of the hospital pharmacist it had been given as a mouth wash, but was now administered internally. The administration of calcium lactate was increased to five grains every hour, on the suggestion of Dr. Thomas B. Wood.

In view of the fact that laboratory tests had shown the clotting time and bleeding time normal, and also that clotting had always occurred promptly on exposure of the blood to the air, I believed the tissue calcium to be at fault, and that some medication was required to immediately alter calcium metabolism in the tissues as distinguished from the blood. A further peculiarity of the tissue reaction was the complete failure of the tissues making up the tonsillar fossa to show the usual post-operative contraction and thickening up to this the ninth day post-operative. Acting on this belief in a fautly tissue calcium metabolism, I ordered hypodermic injection of a soluble thyroid-pituitary-spermin preparation plus five drops of adrenalin, these to be repeated every three hours. In this medication the main reliance was, of course, on the thyroid content of the combination, the others being adjuvant. I wished for the addition of para-thyroid. but knew of no preparation suitable for hypodermic administration that contained this element.

September 2. From now on, because of some of the medication given or because the time was ripe for the hemorrhage to cease, there was rapid improvement. On this date a firm clot 1½ centimeters in diameter was removed from the lower pole of the left tonsil, from which there had been slight hemorrhage. Bleeding was easily controlled and silver nitrate, 50 per cent, applied to the place from which the clot was removed.

September 3. A small, firm clot three millimeters in diameter was removed from the lower pole of the left tonsil. Bleeding was controlled without difficulty.

September 4. No bleeding.

September 5. Coagulation time, partial, 7 minutes; complete, 10 minutes. Blood platelet count 30,000 per cubic millimeter. Hemoglobin 51 per cent.

September 8. Discharged from hospital.

September 13. (Seen in out-patient department)—healing in tonsillar fossa very backward. Patient had cut his finger the previous night and the bleeding was only controlled by wrapping it tightly with an adhesive tape.

September 20. (Seen in out-patient department for last time)—healing in tonsillar fossa not yet complete (28 days post-operative).

September 21. Hemoglobin 52 per cent; red blood cells 2,900,000; blood platelets 177,000; coagulation time five minutes; Wassermann reaction negative, with mass coagulation unusually quick.

December 20. Blood platelet count 59,000.

Discussion: After the hemorrhage was controlled and the patient discharged to the out-patient department considerable clinical and laboratory work was done in order to find something that might have given a warning before the operation, and no concrete fact was developed, except the history of childhood hemorrhages and the low blood platelet count. These and the other facts of childhood history were told by his mother when she came on from the South, after his condition became alarming. The concrete facts on which to base a diagnosis are few, most of the data elicited, even after the discharge of the patient from the hospital, being negative or due to the secondary anemia. The blood platelet count of 30,000 on the 5th of September, increasing to 177,000 on the 21st, and receding to 59,000 on the 20th of December, may have been due to the citrate administered and the citrate transfusions. In the Archives of Surgery for September Brines[2] makes the claim that citrate destroys the blood platelets and so retards the coagulation of the blood. Three facts in this case bear out that claim. First, the blood platelet count was reduced to 30,000, the lowest count recorded, immediately after the administration of citrate and the citrate transfusions; the coagulation time was increased from six to ten minutes, and third, clinically the patient showed an increased tendency to bleed after each transfusion. The diagnostic criteria not due to the secondary anemia and the citrate administered include the following: The umbilical hemorrhage at birth and the other hemorrhages of childhood, pointing to the probability of the condition being congenital; the attack of "rheumatism" at the age of ten; the loose nature of the clots, and the slow healing. The rheumatoid attacks are of no diagnostic value. Against a diagnosis of hemophilia are the absence of a concrete history, the normal bleeding and coagulation time, the character of the clots, the intestinal bleeding, if this was present, and the continued low blood platelet count. Against a diagnosis of purpura are the absence of petechial hemorrhages, the congenital nature of the hemorrhagic diathesis, and the rapid increase in the blood platelet count soon after the last citrate was administered. In my opinion, the most that can be said is that this patient was a sporadic bleeder of undetermined type, possibly a type not yet described specifically.

POINTS OF INTEREST.

First—The patient was neither purpuric nor hemophiliac, the usual types of bleeders, so far as could be determined.

Second—The continued bleeding for eleven days in the presence of a normal coagulation and bleeding time, and the immediate clotting of the blood on exposure to the air.

Third—The complete failure of three transfusions, the specific treatment for purpura and hemophilia, to effect the bleeding, as well as the failure of two intravenous injections of sodium citrate.

Fourth—The continued low blood platelet count in this unusual type of habitual bleeder.

Fifth—The failure of the tissues in and about the tonsillar fossa to contract and thus bring about the desired constriction of the lumen of the blood vessels.

Sixth—The very prolonged healing following the operation.

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223-225 East Seventeenth Street.

AN UNUSUAL CASE OF VINCENT'S ANGINA.

DR. EDWARD R. ROBERTS, Bridgeport, Conn.

It is thought that this case is of sufficient interest to report, not because it is atypical for such a case but because of the severity of an infection usually very lightly treated in text books, namely a case of Vincent's angina which for a time looked like a fatal illness.

Mr. M., while touring in Canada was seized with a very severe sore throat, intense headache and muscular pain, fever and sweating. He was seen by the writer on about the tenth day of his illness with a temperature of 102, and judging from the appearance of the throat and the history it was felt that he had had a simple tonsillitis and perhaps a mild Quinsy, left side, which he had absorbed. The throat was not sore nor was it especially so during the remainder of the illness. Each tonsillar crypt was filled with material characteristic of a simple tonsillitis, in no way suggestive of

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a membrane. Externally a hard swelling the size of a small hen egg was manifest on the left side which was not, but had been, very tender.

Cultures were negative for any definite organism and the case was regarded as a simple one which was much improved in twenty-four hours, as far as the patient's feelings were concerned.

On the second day the patient was, generally, not as well (but with no complaints except exhaustion) and it was thought best to send him to a hospital. His temperature on admission was 105. A few hours later the local manifestations of the disease changed very much in character at which time both tonsils were completely covered with a membrane in which were found the bacillus and spirillum of Vincent's angina. By this time the man seemed very ill although his only complaint still, was weakness, temperature ranging from 102 to 105. Pulse, although not especially rapid, was not of good quality, rate from 112 to 120. Albumen and casts were present in the urine.

The condition of this patient was now such as to warrant any treatment however strenuous and it was decided to give .45 grams of neo-salvarsan intravenously. So far as the writer was at this time aware this drug used intravenously had had a greater effect upon the local disease, and had brought about an improved general condition in this disease more promptly than had any other treatment then recommended. Since Dr. James F. McKernon, New York City, has reported very excellent results in a large series of cases in the A. E. F. with fused chromic acid crystals applied locally. The writer has had no experience with this drug in such concentration.

A blood culture and the Wassermann reaction were both negative.

Unfortunately the salvarsan reaction, considering the patient's condition, was severe, with vomiting and an exhausting diarrhea, so severe in fact, that his condition was considered rather grave.

On the morning following the neo-salvarsan administration the patient experienced a short period of collapse during which cyanosis was marked and the pulse became very weak. Periods of respiratory depression were experienced several times by the patient during the early part of this day, during which he said himself that it did not seem as if he could draw another breath. This condition was not due to any obstruction.

Twenty-four hours after the neo-salvarsan signs of improvement were manifest. The patient appeared better, said he felt

better, and the tonsil membrane was beginning to come away. The pulse and the temperature however remained about the same, falling only by one-tenth to one-fifth of a degree until the morning of the fourth day to a point of 100, but rising to 101.6 in the evening.

On the third day he developed an otitis media on the side of the glandular involvement which necessitated a myringotomy on the fourth. A cover glass preparation of the discharge showed a Type III pneumococcus which was also present in cultures from the mouth, together with the staphlo and streptococcus.

The patient's recollection of the early part of his hospital illness was very hazy, and he had short periods of delirium during that time.

On the fifth day the morning temperature was recorded as 99.8, evening 100, and a pulse around 90 for the day. The membrane had entirely disappeared. Cover glass preparations showing no Vincent organisms. Neo-salvarsan in glycerine was used locally three or four times a day, bismuth and opium to combat the diarrhea and digitalis as a stimulant.

The exhaustion following this experience was marked, even mastication of soft food being very tiring but the rapid return of the appetite was a very striking feature.

This case differs from the usual pharyngeal Vincent's infection because of the severe toxemia, the temperature range, the exhaustion and the complications for a disease usually mild in character. A recent work on the subject is responsible for the statement that kidney complications are unknown.

It is very likely true, that had the patient not had the earlier mixed infection to deal with, he would not have been so overwhelmed. It is also possible that many of the toxic symptoms were dependent upon other organisms, the more especially the third type pneumococcus, but the facts that there was an interval of improvement just preceding the appearance of the Vincent organism, that on their appearance the case developed a very serious aspect, that such immediate results followed the neo-salvarsan administration (a drug known to have a very destructive effect on this type of organism and not upon the others which may have played a part in this infection) it would seem fair to conclude that to the drug, neo-salvarsan, should go the credit for the recovery of this case.

The Wassermann reaction was done as routine in the taking of the blood culture. It is the writer's practice, however, in

each case of Vincent's infection, if at all severe, or if the diagnosis is at all in question to have the blood reaction checked for specific disease, the more especially if it is elected to give an an arsenical preparation intravenously as treatment. This practice was adopted because of personal experience with two other cases and a knowledge of two others in the practice of a syphilologist. It may be of interest to make brief mention of these cases here. The first case, was that of a young man with a destructive (locally) Vincent's. He had been told that he had syphilis and his wife so informed. As a result this innocently acquired illness brought him to the door of the divorce court. The second was a case in which the similarity of the treatment so coincided with the patient's understanding of that instituted for syphilis, and made such a mental impression upon him as to result in a very distressing psycho-motor spastic paraplegia which incapacitated the man for weeks and brought up within the family circle the question of specific disease, causing no end of domestic disturbance. The third case, probably Vincent's, was in a female with the local lesion on the external genitals. It had been diagnosed as syphilis and said to have been checked up by a positive blood examination. A cover glass preparation was not made. After one injection of Salvarsan the local lesion entirely cleared up and the blood Wasserman was persistently negative thereafter. The fourth case was very similar, happening in a male student who was expelled from one of our national institutions (and the entire course of his life changed thereby) because of this pharyngeal lesion.

The writer believes that it is unreasonable to suppose that a syphilitic whose blood had reached the stage of a positive could clear up permanently after an injection of Salvarsan, or on the other hand, that these Vincent infections were responsible for the reported positive Wassermann reactions. One would rather attribute these reports to accidents or errors in technique in taking the blood, labeling the blood or in the actual test.

Whatever the facts the experiences of these patients were of sufficient importance to them to have made a careful checking up of any doubtful point urgently indicated.

THOMPSON: Diseases of Nose and Throat, 1917, p. 415.

THE ROLE OF BACTERIA IN THE PRODUCTION OF ASTHMA AND OTHER ALLERGY.

DR. MARK J. GOTTLIEB, New York, N. Y.

From the Department of Oto-Laryngology at Beth Israel Hospital, New York.

In 1916, Goodale (Boston M. & S. Jour., 1916, 175, 181) reported some preliminary notes on the skin reactions produced by bacterial proteins in vasomotor disturbances of the upper air passages. The organisms used by him at that time were staphyloccus pyogenes albus, aureus and citreus, micrococcus tetragenous, and an organism like the bacillus mucosus capsulatus. The organisms were grown on agar slants and then exposed to ether vapor for four days in order to kill the growth without changing the character of the proteins. The colonies were then washed off with 12 per cent alcohol in water and allowed to stand for one week for the purpose of extraction. The suspensions were used as such for testing skin sensitiveness. Dr. Goodale states that to his knowledge no such work had previously been undertaken. However, in 1917, Gottlieb (6) in a short article on asthma, states that in 1914 he tested one case with glycorinated extracts of pneumococci and stretptococci. To Goodale, however, we are indebted in a great measure for placing bacterial sensitization among the possible factors which cause asthma and other allergic phenomena.

Walker (Jour. Med. Research, 1917, 35, 373) investigated the bacterial flora of the respiratory tract in asthmatics and recovered principally staphylococcus albus and aureus, micrococcus tetragenous and catarrhalis, and bacillus diphtheroidius. He also did agglutination tests with staphylococcus aureus using the serum of 80 patients suffering with asthma. In only three cases was the serum of the patients titrated against organisms isolated from the individual patients. Two of these patients gave agglutination reactions and one was negative. Of the 80, sera tested, 54 showed agglutination, some up to dilutions of 1-250. After reading his work, I had agglutination tests made with the sera of asthmatic patients against the organisms isolated from the nose, throat, teeth, bronchi and stool from each individual case. In each series of tests controls were set up

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CHART No. I.

AGGLUTINATION TESTS.

			Patient's Serum.			Pooled Normal Serum.		
			Dilutions.					
Patients' Initials	Bacteria Isolated	Skin Tests	1–50	1–100	1–250	1–50	1–100	1–250
M. P.	Strep. Vir.	—	neg.	neg.	neg.	neg.	neg.	neg.
	B. coli	+	"	"	"	"	"	"
	Staph. Albus	—	"	"	"	"	"	"
A. D.	B. coli	+	"	"	"	"	"	"
	Staph. Aureus	—	"	"	"	"	"	"
Mrs. S.	B. coli	+	"	"	"	"	"	"
	Staph. Albus	—	"	"	"	"	"	"
	Strep. Hem.	+	"	"	"	"	"	"
	Staph. Aureus	—	"	"	"	"	"	"
G. W.	B. coli	+	"	"	"	"	"	"
	Staph. Albus	—	"	"	"	"	"	"
	Staph. Aureus	—	"	"	"	"	"	"
	Strep. non-hem.	—	"	"	"	"	"	"
C. Y.	Strep. non-hem.	—	"	"	"	"	"	"
	Staph. Albus	—	"	"	"	"	"	"
Mrs. B.	B. Diphth.	—	"	"	"	"	"	"
	Staph. Albus	—	"	"	"	"	"	"
	B. coli	—	"	"	"	"	"	"
M. R.	Strep. hem.	*Stock+	"	"	"	"	"	"
	Staph. Aureus	—	"	"	"	"	"	"
	B. coli	—	"	"	"	"	"	"
I. W.	Strep. Hem.	—	"	"	"	"	"	"
	Staph. Aureus	*Stock++	"	"	"	"	"	"
	Micro. Catarrl.	—	"	"	"	"	"	"
	B. coli	—	"	"	"	"	"	"
L. S.	Strep. non-hem.	++	"	"	"	"	"	"
	Staph. Albus	+++	"	"	"	"	"	"
	B. coli	—	pos.					
S. B.	Gram. + Diplococcus	not	neg.	"	"	"	"	"
	Pneu. iv.	tested	"	"	"	"	"	"
	B. coli		"	"	"	"	"	"
L. G.	B. coli	—	"	"	"	"	"	"
	B. Paratyphoid A	—	"	"	"	"	"	"
R. W.	Strep. non-hem.	—	"	"	"	"	"	"
	Staph. Albus	+	"	"	"	"	"	"
R. W.	B. coli	—	"	"	"	"	"	"
	Gram + Bacillus	—	"	"	"	"	"	"
	B. Diphtheroid	—	"	"	"	"	"	"
M. G.	B. Influenza	—	"	"	"	"	"	"
	Staph. Albus	—	"	"	"	"	"	"
A. S.	Strep. non-hem.	+	"	"	"	"	"	"
	Staph. Albus	—	"	"	"	"	"	"
	B. coli	—	"	"	"	"	"	"
S. S.	Strep. non-hem.	—	"	"	"	"	"	"
	B. Diphtheroid	—	"	"	"	"	"	"
	Staph. Albus	+	"	"	"	"	"	"
	B. coli	+	"	"	"	"	"	"
M. S.	Strep. non-hem.	+	"	"	"	"	"	"
	Staph. Aureus	+	"	"	"	"	"	"
	B. Influenza	—	"	"	"	"	"	"
	B. coli	—	"	"	"	"	"	"
J. K.	Strep. non-hem.	—	"	"	"	"	"	"
	Staph. Albus	—	"	"	"	"	"	"
	B. coli	+	"	"	"	"	"	"
Mrs. R.	Strep. non-hem.	+	"	"	"	"	"	"
	Unidentified Bac.	—	"	"	"	"	"	"
	Staph. Albus	+	"	"	"	"	"	"
	B. coli	—	"	"	"	"	"	"

CHART No. 1—Continued.

AGGLUTINATION TESTS.

Patients' Initials	Bacteria Isolated	Skin Tests	Patient's Serum. Dilutions. 1–50	1–100	1–250	Pooled Normal Serum. Dilutions. 1–50	1–100	1–250
J. K.	B. Mucosus Cap.	+	neg.	neg.	neg.	neg.	neg.	neg.
	Strep. Vir.	—	"	"	"	"	"	"
M. G.	Strep. Hem.	—	"	"	"	"	"	"
Mr. S.	Staph. Aureus	—	"	"	"	"	"	"
	B. Influenza	—	"	"	"	"	"	"
	B. coli	—	"	"	"	"	"	"
L. S.	B. Mucosus Cap.	—	"	"	"	"	"	"
	Gram.—Bac.	+	"	"	"	"	"	"
	Mic. Tetrag.	—	"	"	"	"	"	"
	B. coli	—	"	"	"	"	"	"
	Staph. Aureus	*Stock+	"	"	"	"	"	"
I. A.	Staph. Albus	—	"	"	"	"	"	"
	Strep. non-hem.	—	"	"	"	"	"	"
	B. coli	—	"	"	"	"	"	"
A. F.	Staph. Albus	—	"	"	"	"	"	"
	Strep. non-hem.	—	"	"	"	"	"	"
	B. coli	—	"	"	"	"	"	"
E. H.	Strep. non-hem.	—	"	"	"	"	"	"
	B. Mucosus Cap.	—	"	"	"	"	"	"
	Staph. Aureus	—	"	"	"	"	"	"
	B. coli	—	"	"	"	"	"	"
	Gram. + Coccus	—	"	"	"	"	"	"
Mr. K.	B. coli	not tested	"	"	"	"	"	"
	Strep. non-hem.		"	"	"	"	"	"
	Staph. Auerus		"	"	"	"	"	"
	Staph. Albus		"	"	"	"	"	"
	Strep. Vir.		"	"	"	"	"	"
L. S.	B. coli	—	"	"	"	"	"	"
	Staph. Albus	—	"	"	"	"	"	"
	Gram. + Bac.	+	"	"	"	"	"	"
	Strep. non-hem.	—	"	"	"	"	"	"
	Staph. Aureus	*Stock++	"	"	"	"	"	"
R. G.	Strep. non-hem.	—	"	"	"	"	"	"
	Staph. Albus	—	"	"	"	"	"	"
	B. coli	—	"	"	"	"	"	"
A. F.	Strep. non-hem.	—	"	"	"	"	"	"
	Staph. Albus	—	"	"	"	"	"	"
	B. coli	—	"	"	"	"	"	"

Tests incubated at 37° C. for one hour in water bath, then iced over night and read in the morning.

The amount of bacterial suspension remained constant but the serum dilutions varied. The amount in each tube was 3 cc. and Na Cl solution 0.9% was always added to make up deficiency.

*Stock means that patient reacted only to stock bacteria and not to autogenous.

with pooled sera of non-athmatics. The organisms of 30 cases were used as is shown in the protocol, (chart No. 1). In only one instance was agglutination elicited, although 19 organisms gave positive skin reactions in the individuals from whom they were isolated. The controls were all negative.

In the case where an agglutination reaction was obtained, streptococcus non-hemolyticus was isolated from the teeth and naso-pharynx, staphylococcus albus from the naso-pharynx, and bacillus coli communis from the stool. Her skin reacted negatively to the bacillus coli communis. To streptococcus non-hemolyticus her skin gave a reaction of a central wheal measuring 1x1.5 cm. and a peripheral erythema of 3x3 cm. She also reacted positively to staphylococcus albus; the wheal measured 1x1.5 cm. and the surrounding redness measured 2.75x2.75 cm. This patient's serum agglutinated only the bacillus coli communis isolated from her stool, but she was not found sensitive to this.

From the data published by Walker, one cannot conclude that the agglutination of organisms by sera from asthmatic patients bears a relationship to allergic reactions produced by such germs. Certainly, the evidence presented by me casts a definite doubt upon the assumption that precipitins in the patient's blood are concerned with the production of bacterial allergy, if such a condition exists.

For the purpose of testing asthmatics with bacterial proteins, Walker (Jour. Med. Research, 1917, 25, 487) used a bacterial powder. This was produced by centrifuging bacteria suspended in the saline solution in which they were washed. The sediment was then washed twice in alcohol, then in ether, and then powdered. This was then used on a skin scratch and dissolved in n_{10} sodium hydroxide solution. Three kinds of positive reactions were observed; first, an urticarial wheal; second, a small papule surrounded by erythema, and lastly, simply an erythema. The reaction occurs within thirty minutes. Of 100 cases tested in this way, 67 were negative, and 30 were positive. Ninety-seven were tested with staphylococcus pyogenes aureus, and 19 were found positive in 9 of which the urticarial wheal developed. One hundred and sixty-two cases were tested by me with stock bacterial proteins prepared according to the method described above. The bacterial powder of B. coli, staphylococcus albus and aureus, streptococcus non-hemolyticus, streptococcus hemolyticus, micrococcus catarrhals, pneumococcus, B. diphtheroideus, diplococcus lanceolatus, and micrococcus tetragenous were

used. Eighteen positive reactions were elicited. The cutaneous method of testing was used. Among the 18 positive reactions there was one positive to staphylococcus albus, micrococcus catarrhalis, penumococcus and micrococcus tetragenous; two positive to B. coli, staphylococcus aureus, bacillus diphtheroideus and diplococcus lanceolatus, and 3 positive to streptococcus hemolyticus and non-hemolyticus. In this work, doubtful reactions were disregarded.

It seems to me that this method of testing for bacterial sensitiveness has objections which are valid. The bacterial powder is nothing more or less than dead bacterial bodies—the process of pulverization could not alter the body structure unless they were crushed while frozen in a Buchner press. This evidently is not done. The substances contained in the capsule of the bacteria could only be extracted in a very small measure by the n_{10} sodium hydroxide solution and the serum exuded from the sacrification. Thus only a very small amount of allergic substance can come in contact with the tissues. Furthermore, the process of washing removes practically all of the exogenous bacterial products and it is not known whether it is the endogenous or the exogenous material which causes the reaction.

Contrast the number of positive reactions elicited in the same series of cases using autogenous bacterial suspensions. This is a compilation of 60 cases that gave positive skin reactions to bacteria out of a total of 162 cases tested (chart No. 2). The reactions to stock bacteria are here also tabulated. The bacteria isolated from the various discharges and secretions of each patient were washed from agar slants with normal saline solution and then sedimented by the centrifuge and washed again with saline and again centrifuged. The sediment was suspended in ten times its volume of normal saline solution and then sterilled for one hour each day at between 65° and 70° centigrade for five days. Then each suspension was cultured for sterility and if found sterile used by the intracutaneous method. Normal saline solution was always injected as a control. The 60 cases herein tabulated gave 70 positive reactions with the autogenous suspensions while the stock bacterial powders only gave 17. There were 26 positive to B. coli; 12 positive to staphylococcus albus; 6 to staphylococcus aureus; 1 to streptococcus pyrogenes; 6 to streptococcus non-hemolyticus; 4 to streptococcus hemolyticus; 4 to streptococcus viridans; 1 to an unidentified Gram-negative bacillus; 2 to micrococcus catarrhalis; 1 to bacillus mu-

cosus capsulatus; 5 to pneumococcus; 1 to bacillus influenza, and one positive to bacillus alkaligenes.

The reason why so many positive reactions were obtained with the autogenous organisms as compared with the stock is because during the five days of sterilization, a process of extraction and maceration of the bacteria occurs. Furthermore, with the intracutaneous method of testing the bacterial suspension is deposited between the layers of skin rather than on it and thus a better opportunity for the cells to be exposed to the effects of the exogenous and endogenous bacterial products is afforded.

Two varieties of reaction are observed; one, a swelling at the site of the injection followed by a peripheral hypermia which begins very soon after the injection and reaches its maximum in about 30 minutes; and secondly, a delayed swelling, red and rather extensive, which makes itself manifest usually after six hours. Those that are negative have a slight erythema around the welt produced by the injection which quickly disappears. This also applies to the control. The delayed reaction is never considered, it being construed as a non-specific protein reaction. How much of the immediate reaction should be relegated to the rôle of non-specific protein effect and how much of it can be attributed to specific allergic reaction is not known. For the present, at least, we must be content to wait until the allergen is separated from the rest of the organism and its extract. Very severe reactions were observed and in one instance the effect was of such intensity as to cause necrosis of the skin at the site of the test.

One case gave a severe reaction to B. influenza and is worthy of mention because of its rarity. This patient had had fall hayfever with asthma for about fifteen years, the asthma gradually extending throughout the year but he was always worse at the end of the summer. He was sensitive to ragweed, goldenrod, and potato. He was tested with autogenous suspensions of B. coli, staphylococcus aureus, streptococcus non-hemalyticus, and B. influenza. The last three were isolated from his sputum; the staphylococcus aureus, streptococcus non-hemolyticus, and B. the stool. He gave negative reactions to all except the B. influenza which was severe. On bronchoscopy, the mucous membrane of the bronchi was found to have a granular appearance and bled on the slightest manipulation. After bronchoscopy, he was free from symptoms for about four days. He received no benefit from four injections of a vaccine containing B. influenza and staphylococcus aureus. The aureus was added for its non-

CHART

AUTOGENOUS ORGANISMS.

Case	B. coli	Staph. albus	Staph. aureus	Strep. Pyrog.	Strep. Non-Hem.	Strep. Hem.	Strep. Vir.	Gram. Neg. Bac.	Micro. Catarr.	B. Muc. Cap.
H. G.	+	+								
L. L.	++	+								
C. C.	++	+								
J. M.	+									
M. Mc	++									
B. J.	+++									
K. D.	++++									
R. B.	+									
J. L.	++									
J. G.	+									
E. S.	+									
J. S.	+									
A. J. G.	+									
C. S.	+									
A. W. M.	+									
M. L.										+
J. H.		++								
B. S.		+								
S. Mc	++									
I. W.										
I. M.			++							
M. B.			+							
I. E.		+++	++							
H. B.				+					+	
J. C.	+									+
L. S.								+		
A. M.			+			+				
E. L.	+++						+			
F. L.										
V. T.										
M. S.			+		+					
M. P.	+				+					
D. R.					+					
S. S.			+		+					
W. R.						++			++	
M. B.		+					++			
L. G.										
R. W.		+								
A. M. B.	+									
C. Y.										
M. R.										
A. D.	+									
H. T.										
M. W. G.							+++			
S. S.										
Dr. B.							++			
J. P.		+								
B. I.	++									
T. H.	++									
B. D.		+								
E. B.	+									
S. K.										
C. B.	++									
J. W. S.	+					+				
A. S.					+					
J. D. F.		+								
L. S.		+++			++					
G. W. W.	+									
D. B.										
M. C. B.						++				

— is negative.
+ slightly positive or positive.
++ moderately positive.
+++ strongly positive.
++++ very strongly positive.

Stock tests were made by the cutaneous method.
Autogenous tests were made by the intracutaneous method.

CHART No. II.

STOCK ORGANISMS.

o.	B. Muc.		B.	B.	B.	Staph.	Staph.	Strep.	Strep.	Micro.	Pneu.	Bac.	Diplo.	Micro
rr.	Cap.	Pneu.	Influe.	Alkal.	coli	albus	aureus	Non-Hem.	Hem.	Catarr.		Diphth.	Lanc.	Tetrag.

specific protein effects. He then disappeared, and returned a few years later, complaining in the same old way.

Some years ago, the author was presented with a problem in the form of a case of asthma in which the final analysis of the case led to the intestinal tract in search of a cause of his symptoms. A short resume of the case will explain the idea better than generalizations. Case B. I., 24 years of age, student. During adolescence, suffered a good deal with asthma especially after horseback riding and after playing with the house cat. He was found to be very sensitive to horse dandruff and slightly sensitive to cat hair and ragweed pollen. The house cat was banished and horseback riding was interdicted. He was given a series of treatments with horse dandruff extract and his symptoms disappeared, and he was able to ride again in comfort. The first series of treatments were given during his eighteenth year and he remained free from symptoms until the spring of the following year when he was seized with very severe, constant asthma which necessitated him to leave school and to return to New York for treatment. At this time he was only slightly sensitive to horse dandruff, cat hair and ragweed pollen and it was felt that the severity of the symptoms had to be explained in some other way. He was tested with every known substance that could cause asthma, including all of the trees that pollenated in the early spring but nothing was found. Without much faith and very little hope, I had his stool examined and nothing was found except the usual growth of B. coli. A very thick suspension was dropped on a scarification in his skin and within a very short time a typical large wheal surrounded by a large zone of erythema developed while the contols remained negative. A series of gradually increasing doses of B. coli vaccine put a stop to all of his symptoms. Since then he has had several attacks of asthma due to a return of his horse dandruff sensitiveness which was promptly abated by treatment or he inadvertently played with a cat or was subjected to severe weather conditions. At the age of twenty-two his stool was again cultured and the colon bacillus recovered produced no reaction even though the test was made intracutaneously.

Because of the findings and the result of the above case, I have never omitted having the stool cultured in any case that presented itself for treatment. As has no doubt been noticed in chart No. 2, 26 cases gave positive reactions to colon bacillus; in fact, the number of positive colon bacillus reactions is greater by far than any of the other organisms. This particular phase

of the subject is original with me, and I therefore take the trouble to analyze the 20 cases with a few other related histories for the purpose of placing the evidence before the reader and let him judge for himself whether or not this new field for search after the possible etiological factors in asthma and other allergic phenomena will bear fruit.

Miss A. D., referred by Dr. L. M. Hurd; 35 years of age. First seen on October 17, 1919. For the past three years has been suffering at intervals with profuse nasal discharge accompanied by sneezing, particularly in the morning after arising. Five years previously had some intestinal trouble. One sister had asthma. She was found slightly sensitive to pneumococcus type I, stock. To an autogenous suspension of colon bacillus isolated from the stool, she gave a slight reaction. A vaccine of colon bacillus was injected in gradually increasing doses, and the local reaction was very severe in each instance. During the treatment her condition remained about the same, but about two and one half months after the treatment was finished, she reported to Dr. Hurd that her symptoms were entirely gone.

Mrs. C. B., referred by Dr. Henry Roth; 32 years of age. Appeared for examination the first time on May 22, 1920. For the past four years has had attacks of shortness of breath, cough and expectoration. Mother had asthma. She gives a moderately positive reaction to chicken feathers, slightly positive reaction to pea, and crab; moderately positive reaction to ragweed pollen, and slightly moderate reaction to rye, yellow daisy and orchard grass pollens. She gave a moderately severe reaction to an autogenous suspension of colon bacillus isolated from the stool. Chicken feathers were eliminated from her environment. A series of colon bacillus vaccine injections were given and the patient has since been entirely well, except for a very mild attack of hay-fever each fall. In this case it is difficult to state which of the two measures instituted was instrumental in producing the result obtained, or whether the effect of the combined treatment might not have produced the result.

Mrs. J. L., referred by Dr. S. Zuckerman; 32 years of age. Was first seen on November 20, 1920. Since the middle of June, 1920, has had attacks of sneezing. This occurs usually as soon as she places her foot on the floor as she is arising in the morning. For the past three weeks, the skin in and about the nose has itched continuously. Had been constipated for about two months. She has had eczema since childhood. About one year ago, had an attack of urticaria with which her eyelids and

lips were swollen. She is not sensitive to any of the substances used for testing, with the exception of goldenrod, ragweed, and yellow daisy. A moderately severe positive reaction was obtained with an autogenous suspension of colon bacillus isolated from the stool. She was given a series of treatments with colon bacillus vaccine and within a short time her symptoms subsided, and except for mild hay-fever during July and August, feels entirely well.

Miss E. L., referred by Dr. M. Sher; 19 years of age. First appeared for examination on February 19, 1921. Her chief complaints were obstructed nasal respiration, sneezing and watery discharge of the nose. Her maternal uncle has hay-fever. Suffers with constipation. Her present condition began in March, 1920. The symptoms occur mostly in the morning while getting out of bed. Is compelled to sleep with her mouth open. She is slightly sensitive to chicken feathers. The uric acid content of her blood is 2.4 mgm. per hundred cc. She was slightly sensitive to an autogenous suspension of streptococcus viridans isolated from the naso-pharynx, and strongly sensitive to colon bacillus isolated from the stool. She has a high deviation of the septum, to the right. A series of vaccine treatments containing streptococcus viridans and colon bacillus was given, and although her symptoms diminished considerably, they did not entirely disappear. The uric acid content of the blood being higher than the average normal, all food containing large quantities of nucleinic acid was interdicted, and the feather pillows on her bed were changed to floss pillows. Even with these factors given their proper attention, the symptoms continued and her nasal septum was resected and then the symptoms abated completely.

Mrs. A. J. G., referred by Dr. L. M. Hurd; 52 years of age. Was first seen on June 8, 1920. Her chief complaint was sneezing, nasal discharge, and obstructed breathing since the age of forty-one. Three or four months ago, began to have asthmatic symptoms. For the past seven years, has had an itchy rash in the hair at the back of the head, eyes also itch. Is habitually constipated. Father had asthma. She gave a moderately positive reaction to cabbage, and slightly positive reactions to corn, oat, and wheat leucosin. An autogenous suspension of colon bacillus isolated from the stool produced a slightly positive reaction. A series of colon bacillus vaccine treatments were given by Dr. Hurd. This patient has now been without symptoms for about two years after the treatment was given. The beneficial

effects were felt two months after the vaccine had been administered.

Mr. M. P., referred by Dr. M. Barshak; 23 years of age. He consulted me for examination and treatment on October 15, 1919. Chief complaint was shortness of breath which has lasted for six weeks. The patient feels well while at home but when he visits other houses, becomes ill. Is constipated during attacks. An abscess was found at the apex of the left upper central incisor and this was removed. Uric acid in the blood was found to be 2.7 mgm. per hundred cc. He was slightly sensitive to chicken feathers and an autogenous suspension of streptococcus non-hemolyticus and B. coli. Because of the moderate increase of uric acid above the normal, parenchymatous foods were forbidden in his diet, and alkaline waters in large quantities were given. In addition to this, a vaccine containing streptococcus non-hemolyticus and colon bacillus were given. Except for very mild attacks which have occurred only when he is traveling, he has felt well since.

Mr. J. C., 53 years of age; appeared first for treatment on November 7, 1919. For the past five years, has complained of shortness of breath, wheezing, cough and expectoration. Worse at night. Constipated. Has pyorrhea alveolus. Gives a slightly positive reaction to an autogenous suspension of B. coli. Since his examination he has not returned for treatment.

Mrs. L. L., 44 years of age; first appeared for examination and treatment May 1, 1922. Her chief complaint was cough and wheezing for the past twenty years. For the last three years, has had outspoken attacks of asthma. She is not sensitive to any proteins to which she was tested. She gives a positive reaction to autogenous suspensions of colon bacillus and a slightly positive reaction to staphylococcus albus. A combined vaccine containing staphylococcus albus and B. coli was given in gradually increasing doses, and for a period of about six months, showed marked improvement, then relapsed into her former condition. A second series of vaccine injections is contemplated.

Mrs. A. W., referred by Dr. L. M. Hurd; 43 years of age. First appeared for examination on October 25, 1920. For the past five months had a severe attack of vasomotor rhinitis. She is slightly sensitive to peach and to autogenous suspensions of B. coli. A series of vaccine treatments were given and no improvement obtained.

Miss M. M., referred by Dr. L. M. Hurd; 38 years of age. Presented herself the first time for examination, October 25, 1920.

Her symptoms were essentially vasomotor rhinitis which she had for the past six months. She was not sensitive to any of the substances to which she was tested, with the exception that she was moderately sensitive to an autogenous suspension of B. coli. A series of vaccine injections containing colon bacillus gave her no relief.

Miss B. J., referred by Dr. Chas. Jurist; 21 years of age. For the past eight months has had what is essentially vasomotor rhinitis. She is slightly sensitive to almonds, and severely sensitive to colon bacillus from her stool. A vaccine containing colon bacillus was given by her physician, and the following letter which was written on March 26, 1922, is self-explanatory:

Dr. Mark J. Gottlieb,

Dear Doctor:

In reply to your letter of the 23rd, I wish to inform you that two months after the completion of a series of injections of the vaccine prepared by you, complete relief was obtained. She remained entirely free from symptoms for six months, when there was a recurrence. I then had a local laboratory prepare a fresh vaccine from her stool and after the injections were completed she felt entirely well and has remained so up to this present writing.

With many thanks for your kindness and interest, I am

Sincerely, Dr. C. Jurist.

Cases S. J., C. C., R. B., C. S., and J. M., were sensitive to colon bacillus, but have not responded to inquiry by letter as to their condition, and therefore, no report can be made.

Mr. G. W. W., referred by Dr. John McCoy; 15 years of age. First appeared for examination on October 24, 1919. For the past eight years, sneezes when he goes out of doors. During the past five weeks, has had repeated attacks of asthma. He was found to be slightly sensitive to cheese, pork, dog hair, coffee, peanut, and moderately sensitive to corn, and severely sensitive to ragweed. Streptococucs non-hemolyticus and staphylacoccus aureus were isolated from his nose; B. coli and an unidentified gram-positive coccus were isolated from the stool. He was found to be slightly sensitive to B. coli isolated from his stool, and negative to the other organisms. This patient did very well under a rigid diet and avoidance of dogs, and an autogenous vaccine made with colon bacillus. Whether, or not, an abstainance of the foods to which he was sensitive alone would have produced this result, is impossible to state.

Mr. J. G., referred by Dr. J. Singer; 35 years of age. First appeared for examination on October 8, 1920. This is essentially a case of fall hay-fever, with sneezing during the entire year. He is moderately sensitive to cat hair, slightly sensitive to apple, coffee, and lettuce, very strongly sensitive to goldenrod and yellow daisy, and moderately sensitive to ragweed. There is a cat in the house. From his tonsils, streptococcus and staphylococcus aureus were isolated; from his nose and teeth, staphylococcus aureus, and from the stool, B. coli and staphylococcus aureus. He was found very slightly sensitive to an autogenous suspension of B. coli, and negative to the other organisms. He refused to have vaccine treatment, and no improvement was noted after getting rid of the cat.

Mrs. E. S., referred by Dr. H. M. Weissman; 29 years of age. Presented herself first for examination on July 19, 1921. About four months before, developed nasal discharge, cough, expectoration and dyspnea which have continued up to date. She was not sensitive to epidemal, stock bacteria, food, or pollen proteins. She was found to be slightly sensitive to an autogenous suspension of B. coli. She did not finish the B. coli vaccine treatment, and no improvement was noted.

Miss H. G., referred by Dr. I. H. Goldberger; 33 years of age. First appeared for examination and treatment on March 14, 1922. This is essentially a case of horse dandruff asthma. She was also found to be slightly sensitive to an autogenous suspension of staphylococcus albus and moderately sensitive to B. coli. She received a great many treatments with horse dandruff extracts, and her symptoms subsided considerably, but not entirely. The sensitiveness to horse dandruff which was severe at the start was found to be slight at the end of treatment. Colon bacillus vaccine treatment was not given.

Mrs. S. McH., referred by Dr. John McCoy; 45 years of age. Presented herself first for examination on December 15, 1920. Her chief complaints are sneezing and running of nose for the past five weeks. Worse at night. Sunlight is disturbing to the patient. She was not sensitive to any of the proteins to which she was tested. From her stool and nose, pneumococcus was isolated, and from the stool, B. coli. She was found to be slightly sensitive to an autogenous suspension of pneumococcus, and moderately sensitive to B. coli. A series of vaccine injections with both these organisms was given, but no result was obtained, and subsequently high colonic irrigations were given. This kept the patient free from symptoms for about six months,

and then the annoying condition returned, and subsided after further intestinal treatment.

Mrs. J. W. S., referred by Dr. John McCoy; 31 years of age. Appeared for examination on October 16, 1919. Her chief complaint was obstructed nasal breathing for the past six months, especially when she lies down. Sneezes considerably, and the nose becomes obstructed, developes difficult breathing and wheezing. Expectorates yellow sputum. For the past six years, has had some nasal trouble consisting of chronic ethmoiditis with polypi. This entire condition developed after a nasal operation eight months ago. She was found to be slightly sensitive to corn. Streptococcus hemolyticus and staphylococcus aureus were isolated from the nasal discharge, teeth and sputum, staphylococcus albus from sputum and stool, and B. coli from the stool. She gives a slightly positive reaction to an autogenous suspension of streptococcus hemolyticus and B. coli. A vaccine containing streptococcus hemolyticus and B. coli was given and the result from this is not known because it is impossible to get into communication with the patient.

The following three cases are ones of urticaria and eczema which have an intestinal basis for their being, as will be shown in the histories.

Miss K. D., referred by Dr. S. Kemper; 23 years of age. Appeared first for examination on February 17, 1922. For the past six months, has had an itchy rash of the body and face, occasionally it is absent. Three years ago suffered with sneezing and itching of the nose. This has disappeared. She was not sensitive to any of the substances to which she was tested. She was strongly sensitive to an autogenous suspension of colon bacillus. After receiving a series of vaccine treatments, the rash was only present at times and the skin reaction of an autogenous suspension of the same organism from her stool gave only a moderate reaction. She was then given another series of vaccine injections when the entire rash disappeared and she has been well since.

Mrs. E. B., referred by Dr. Eli Moschowitz; 53 years of age. First appeared for examination on October 27, 1921. Since March, 1921, has been suffering with hives. This occurs after the slightest irritation, especially after undressing at night. She is slightly sensitive to cantaloupe, and to an autogenous suspension of colon bacillus. She refused to be treated by vaccine and in their stead she was instructed to take three cakes of yeast a day, and after partaking of this for a considerable time, the rash entirely disappeared.

Mrs. T. H., 34 years of age. First came for examination and treatment on February 11, 1921. For the past few years she has suffered with eczema at the back of the neck below the hair line, and itching of the perineum. She was not found to be sensitive to any of the proteins to which she was tested. She was moderately sensitive to an autogenous suspension of colon bacillus from her stool. After a series of vaccine injections containing B. coli, the condition disappeared, and she has continued to be well up to date.

As shown in the three cases depicted above, the intestine has a very definite bearing upon the production of so called allergic manifestations in the skin. The following two cases are interesting and instructive, not because the patients were sensitive to colon bacillus, but on account of the fact that they were sensitive to foods, and the bacteriological findings of the stool are greatly at variance with the usual findings.

Mr. J. B. K., 38 years of age. First appeared for examination and treatment on March 25, 1920. For the past seven years, has had hives and stiffness of muscles with pain on movement, has difficulty in going to sleep, and when he does sleep, it is very heavy. On arising in the morning, he feels dull, apathetic, and disinclined to move on account of stiffness and pains in the limbs. The uric acid content of his blood was found to be 7 mgm. per hundred cc. He is sensitive to pork, crab, cabbage, cauliflower, banana, strawberry, rye and tobacco. The ordinary colon bacillus that is usually found in the stool of most people was practically replaced by a gram-positive unidentified coccus. He was not found sensitive to either of these organisms. Because of the high uric acid content of his blood, he was instructed not to eat substances containing an abundance of nuclei and to abstain from partaking of the foods to which he was sensitive. He was given a series of vaccine treatments containing the gram-positive coccus and B. coli. In addition to this, every week, one quart of a bouillon culture of colon bacillus was slowly instilled into his lower bowel. The patient himself summarizes the effect of treatment, as follows: before treatment, attacks of hives; subcutaneous swelling of tissues; stiffness in bones and joints; difficulty in getting to sleep; extreme heaviness of sleep; waking in the morning, heavy and dopey; tenseness of muscles and desire for action; craving for sweets after meals, (eats two to three pounds of candy, weekly). After treatment was instituted, no hives, swellings or stiffness; sleeps quickly, and from ten to twelve hours; sleep is not as heavy;

on awakening, his wits are clear; there is no tenseness in his muscles; has no desire for sweets or even sugar. After treatment was completed, the uric acid content of his blood was found to be 4.5 mgm. per hundred cc. This, of course, is still high, but with care in diet and continuous observation, this patient has been enabled to live comfortably without his previous suffering.

Mrs. E. H., referred by Dr. John McCoy; 60 years of age. Applied for examination on January 3, 1920. For the past 30 years, has had repeated attacks of loss of voice, and wheezing. Has a very slight cough and occasional attacks of discharge from the nose. Is worse in a close room and during damp weather. Suffers with an itchy rash in the scalp after partaking of shell fish. Gives a very strongly positive reaction to peanut, strongly positive reaction to barley, moderately positive reactions to rice, corn and wheat gliadin, and slightly positive reactions to orange, strawberry, bean and whole wheat. From the nose and throat, staphylococcus albus, streptococcus non-hemolyticus and bacillus mucosus capsulatus were isolated. Bacillus coli communis was practically replaced by an unidentified gram-positive coccus in the stool. She was not found sensitive to autogenous suspensions of these organisms. All of the foods to which she was sensitive were eliminated within practical limits from her diet, and a vaccine containing the unidentified gram-positive was given. This patient did very well, all of her symptoms having disappeared.

The two cases cited above and a few others in my files suggest the idea that a variation from the normal intestinal flora may operate to render the intestinal mucous membrane easily permeable to undigested food proteins, which when they gain access to the tissues produce symptoms because the individual has an idiosyncracy to them or the abnormal intestinal organisms may have the power of so altring the proteins as to render them easily absorbable and in such chemical form that the body cells are readily affected by them. An investigation along this particular line, I am sure would be productive of interesting and useful facts.

Excluding the last two cases because no sensitiveness was demonstrated to the bacteria isolated from the stool, there were twenty-six cases in which definite sensitiveness to B. coli was elicited. Twelve of these cases were definitely free from symptoms after treatment. The few others who showed improvement are not considered. Whether or not, the other measures insti-

tuted in the treatment were entirely responsible for the results cannot be definitely stated, it is an interesting fact that among a great many of the cases herein reported who received no benefit, insufficient or no treatment was given with the colon bacillus vaccine. Whether the effect of the B. coli vaccine is purely a non-specific protein reaction or a combination with specific effect is impossible to say. My personal belief is that it is a combination of both, because very severe local reactions are usually experienced from the vaccine. The matter of sensitiveness to colon bacillus was mentioned by me in two previous articles, first in 1920 (Jour. A. M. A., 1920, 74, 14, p. 931), and later in 1921 (Laryngoscope 1921, XXXI, 12, p. 957).

It is contended by Cooke (J. Immunology, 1922, 7, p. 147), that bacterial allergy has not been proven to exist because of the fact that very often reactions are obtained with stock organisms but similar organisms are not found in the cultured material from the respiratory tract. The respiratory tract is not the only place in the body that can harbor organisms the absorption of the products of which are liable to produce allergic phenomena. The intestinal tract is a frequent source of such absorption. When one considers that the gall bladder, or the appendix may be chronically diseased, and that abscesses at the apices of teeth are very often carried for years without the individual being cognizant of it, it is unfair to assume that because the organism sought is not found in the respiratory tract, that the finding of sensitiveness to a stock organism has no basis, in fact. Furthermore, the patient may have had an infection, such as a furuncle or carbuncle, and become sensitive to the germs producing the infection and yet no longer be a host for that particular variety of bacteria. The same author insists that because of the fact that no account has come to his attention of a patient being shocked by an overdose of a bacterial suspension that it is not possible to accomplish this. My firm opinion is that the amount of allergic substance accompanying bacteria is very small and to produce this effect, the dose must be so large that the effect of the bacterial toxins and non-specific protein action would be so great as to either kill or prostrate the patient for a long period. I do not believe that this particular point is susceptible of proof unless the component parts of bacteria and their exogenous products be separated from each other and tested individually.

43 West 69th Street.

PHILADELPHIA LARYNGOLOGICAL SOCIETY.

Stated Meeting, October 9, 1923.

A stated meeting of the Philadelphia Laryngological Society was held on Tuesday evening, October 9, 1923.

The following papers were presented:

"Acriflavine. Its History and Use in Aural Surgery." Dr. Jas. H. Mendel.

"Nasal Neurosis" (Accepted Candidate's Thesis of the American Laryngological, Rhinological and Otological Society). Dr. Robert F. Ridpath.

"An Interesting History of a Patient with Sphenoidal Sinus Disease." (Presentation of Patient.) Dr. Philip S. Stout.

"Cyst of the Epiglottis" and "Report of a Case of Abscess of Lingual Tonsil." Dr. J. Leo Donahue.

"A Preliminary Report of a Case of Peculiar Tumefaction Involving the Left Parieto-Frontal Region." Dr. Wm. G. Shemeley.

An Interesting History of a Patient with Sphenoidal Sinus Disease. (Presentation of Patient.) Dr. Philip S. Stout.

Report of Case: Abstracted.

History: A woman, age 41, single, saleswoman. Complaining of periodic headache on top of head for past ten years, associated with a profuse purulent discharge from nose for the same duration. Family history negative, except, one brother died at the age of twenty-nine, as a result of a severe hemorrhage from his lungs. Past personal history negative, except, she contracted a syphilitic infection in 1917, for which she received, immediate, active treatment and obtained a negative Wassermann within a year's time.

Present condition started in 1913, when she began to suffer with frequent attacks of cold in head associated with a profuse purulent discharge from nose. During the attacks the nose would first be stuffy, then later there was a mucoid discharge which was highly irritating, which became purulent, necessitating the use of numerous handkerchiefs daily, associated with this, there was a severe headache, which was principally on the top of her head, covering an area of 6x4 c.m., which if pressed would make her blind, giddy and nauseated. The pain would radiate to both ears, then to the shoulders and finally go down her spine. Has a constant dull ache between the eyes also behind the same, which feels as if something heavy and solid were there. States that her vision has been falling off rapidly.

Upon the first visit to my office patient states that she could hardly see me, and only very indistinctly, her face was stiff and expressionless, was complaining of pain throughout the body; besides very severe pain in her head, had sensations of being cold and chilly although the day was warm. Had not a good night sleep in months, feared she was losing her mind. As soon as she would lie down, her head would throb violently and she would break out in a profuse perspiration. In the morning her joints and muscles would be so sore and stiff that she could hardly move. So for ten years she suffered this way with very little relief. Every physician in her town had been called in attendance, but none was ever able to make a correct diagnosis or give her much relief. Many of them attributed her trouble to her syphilitic infection,

although she had manifested the same symptoms four years prior to receiving her initial lesion.

September 4, she came to Philadelphia and was treated in a nerve clinic for six weeks without any relief; while in the dispensary she took the initiative and visited the nose and throat clinic and it was there that the diagnosis was made—but! the physician in charge made a great error in wanting to operate immediately, so frightened was she—that she left the clinic and came to my office an hour later.

The examination of her nose revealed the following: Nasal mucous membrane and turbinates shrunken, (previously done at nose and throat dispensary) so that with a pharyngoscope I saw a picture that could never be forgotten—namely, two lines of pus were making their exit, bow-like from each sphenoid ostium and running together on the posterior wall of the pharynx. The posterior group of ethmoids were envolved, as evidenced by the presence of pus coming down from their respective ostii. By transillumination the frontals and maxillary sinuses were negative.

X-ray findings showed pus in the sphenoids and possibly in posterior ethmoids.

Eye findings are as follows: Right eye 20/75, Jaeger 7. Left eye 20/75, Jaeger 8. There is evidence of an old posterior synechia in each eye. Right eye-media clear, except a few spots of plastic exudate on anterior capsule, disc oval at 90, margins poorly defined, cupping central, shallow, blood vessels full, macula and periphery negative.

Left eye same as right.

Another eye examination October 3, one month later, reveals as follows: Right eye 20/40, Jaeger 6. Left eye 20/40, Jaeger 6. Nerve head and retina show less edema. Wassermann, negative.

Treatment: Suction with fifteen pounds negative pressure was used for twenty minutes every other day. By this method I was able to evacuate a considerable amount of pus. This was followed by tamponage of silver albuminate solution.

After five weeks of treatment recovered sufficiently to go back to work.

DISCUSSION.

DR. RIDPATH: There are numerous cases of sphenoiditis that are undiagnosed. Every case of headache that is presented to you should receive the most careful consideration in reference to determine its source. The use of the pharyngoscope has a very practical value in making a diagnosis of sphenoiditis.

....DR. STOUT, closing: I wish to lay particular emphasis on the carelessness of the numerous physicians who attended this patient. Not one of them made an intranasal examination or referred her to a nose and throat specialist and furthermore the value of suction which I think is indispensable in these cases.

Case Report—Abscess Lingual Tonsil. Dr. John L. Donahue, Philadelphia, Pa.

Patient, G. C. G. Examination, 9/23/23. Claimant of the U. S. Veterans' Bureau was seen on the above date and complained of the following symptoms: sore throat, dysphagia, profuse salivation, sense of pressure in the throat, and marked alteration of voice.

The patient revealed upon examination the following: mouth negative, naso-pharynx negative, faucial tonsils completely removed, the lingual tonsil reveals an inflammatory area on the left side covered with minute whitish spots, there is some edema about this area. External examination of the throat shows marked swelling on the left side with glandular involvement. Examination on 9/25/23, shows an abscess like mass of the left lingual tonsil with all of the above symptoms markedly increased. The patient was hospitalized at the U. S. Naval Hospital, Philadelphia, this date, and late in the afternoon there was a spontaneous rupture of the abscess mass with immediate relief of all the symptoms.

Patient was seen about four or five days later and only a slight subacute inflammatory condition of the lingual tonsil remained.

Cysts of Epiglottis. Dr. John L. Donahue, Philadelphia, Pa.

Patient, J. V. M., age 34, color white.

Examination date, March 28, 1923. Family history, negative. Previous personal history: Gassed (phosgene and chlorine), September 26, 1918, from which a frontal sinusitis developed November, 1918.

Present Complaint: Pain over the frontal region, watery discharge from nose, cough and expectoration.

Examination: Septum deflected to the left and in contact with large left middle turbinate bone. Tonsils are infected, there is hypertrophy of the lingual tonsil. Over the anterior surface left side of epiglottis, a small mass, semi-globular in shape, red in color, smooth, pitting on pressure, broad based, is seen. Subsequent examinations showed alteration in the size of this mass.

X-ray of the sinus was negative. Transillumination was negative.

Diagnosis: Cyst of the epiglottis based upon the following points: situation, color, smooth semi-globular surface, pitting on pressure, decrease in size of tumor.

This tumor was not aspirated because it was not causing any symptoms and the patient did not wish to have anything done to him; he was unaware anything abnormal was present in his throat.

Etiology: Due to retention from obstruction of the muco-parous gland or distention of the lymphatic vessel. Aspiration of such cysts often shows T. B. bacilli and giant cells in the content. The treatment is to remove it surgically or with the galvano-cautery, if symptoms are present.

The following tumors found in this situation are lipoma, angioma, fibroma, papilloma, enchondroma, lymphoma, myxoma.

Differential Diagnosis: Lipoma—yellow or pink in color, large and solitary, broad based or pedunculated, movable, smooth lobulated, elastic to probe and very rarely found on the epiglottis.

Angioma—very rare, single, sessile, very irregular, varies in size, bright or dark red, found on vocal cords, ventricular bands, aryepiglottic folds, or sinus pyriformis, not found on the epiglottis proper.

Fibroma—found on the vocal cords as a rule, rarely found on the epiglottis, they occur single and sessile, embedded or pedunculated; their color is grayish, white, pink, or dark red, growth is rounded and generally smooth, but may have a mulberry surface when they are termed a papillary fibroma; they vary in size from a pea to an acorn.

Papilloma—rarely found on the epiglottis. Papilloma does not invade the tissue from which it springs, color is white, bright red or pinkish, single or multiple, may be broad based or pedunculated and heaped up in a cauliflower mass, surface is irregular or watery.

Frequency of Cyst: Moritz Schmidt reports eight cysts, two in men and six in women, during ten years service at his clinic, from 32,997 patients examined. The condition is more interesting than troublesome.

December 4, 1923.

Case Presentation: Pneumococcic Meningitis, following Otitis Media with Apparent Recovery, followed by an Obscured Intracranial Complication. Dr. Henry S. Weider.

(To appear in subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. GEORGE W. MACKENZIE said this interesting case deserved some discussion. Before performing any operation a complete diagnosis ought to be made, particularly so in cases of operation upon the mastoid. From the report of this case it did not appear that any attempt was made to examine the hearing or vestibular functions prior to September 17, about a month after the operation on the mastoid, in spite of the fact that the patient presented two important signs referrable to the inner ear, staggering gait and marked spontaneous nystagmus, rotary, toward

the left side on admission to the hospital. Nystagmus to the left, I. B., away from the affected side, is a symptom characteristic of labyrinth destruction and not of labyrinth irritation, as the essayist appears to think. Nystagmus toward the affected side speaks for a labyrinth irritation. Furthermore, the labyrinth findings of September 17, again on October 5 and on October 26, all speak of labyrinthine destruction, which in the presence of the middle ear suppuration is recognized as a diffuse labyrinthine suppuration, and as such it ought to have been recognized early and operated accordingly; that is, thorough labyrinth drainage.

Dr. Mackenzie then suggested that they review the findings of the case as presented by the essayist, to see what there was to support the diagnosis of labyrinthine suppuration. When the patient was first seen, there was (1) staggering gait; (2) rotary nystagmus away from the side of the suppurating ear; (3) headache; (4) nausea and vomiting. There was no reference made to the hearing function from the patient's viewpoint and unfortunately no record of tests having been made until more than a month after the first operation when the tests as recorded, were —right ear, air conduction, absent for the C, 128, fork, but the patient claimed that the bone conduction was heard in the ear, and Webster referred to the (unaffected) left side. No more definite test was made to determine absolute deafness. As far as the findings go they suggest destruction of the perceiving apparatus, for the presence of slight bone conduction is always present in such cases, due to the borrowing of bone from the opposite, intact side.

Further evidence corroborative of the diagnosis of labyrinthine destruction (suppuration) was presented in the report by the turning test findings as follows: About the middle of September, the after-turning nystagmus to the right was ten seconds, and to the left sixteen seconds. On October 5, the after-turning nystagmus to the right were twelve seconds and to the left eight seconds. On the same date the caloric test was made on the right ear and found to be negative after four minutes. On October 26, the after-turning nystagmus to the right was six seconds and to the left seventeen seconds. These were very characteristic findings of labyrinth destruction, when one considers that the normal after-turning nystagmus to each side, after ten turns is twenty-four seconds, which added together make forty-eight seconds for both sides. In the event of destruction of one side, the sum total of figures to the two sides drop to one-half of forty-eight or twenty-four seconds. In other words, if one added the duration of the after-turning nystagmus to the unaffected side of sixteen seconds, twenty-four seconds would be obtained, which is the average findings for a recent case of labyrinthine suppuration. If one strikes an average of the sum total of the figures, on these tests made in the case under discussion, they equal sixty-nine seconds, which is three less than seventy-two seconds, which is the average sum total for the test. Again, if one adds together the three test findings to the left (sixteen plus twelve, plus seventeen), forty-five seconds would be obtained. If this were divided by three to obtain an average, one would get fifteen seconds, which is mighty close to sixteen seconds. Again, if one adds the duration of the three findings to the right (six plus eight, plus ten), they equal twenty-four, which divided by three gives eight seconds, which corresponded exactly Dr. MacKenzie contended as the characteristic findings for after-turning nystagmus toward the diseased side.

He thought the essayist had reported a case which was undeniably that of labyrinthine suppuration. Furthermore, the suppurative inflammation of the inner ear is responsible for the symptom referable to the cerebellum, the strongest evidence of a cerebellar lesion is the reversal of the nystagmus from the left to the right side, indication an irritative lesion, which may later on swing over to the left again if the irritative stage of inflammation in the posterior fossa should pass over into the suppurative stage. He thought the case a most interesting one and that the Society would be pleased to learn of the future turn of events.

Dr. Weider in closing said the suggestion that Dr. Mackenzie had made received serious consideration. Personally, he was much in favor of it.

A Case of Advanced Tuberculosis of the Larynx Arrested by Intralaryngeal Injections of Chaulmoogra Oil. Presentation of the Patient. Dr. Robert M. Lukens.

(To appear in subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. HERMAN COHEN asked if any cocain was given before the treatment.

DR. F. M. STROUSE wished to know if chaulmoogra oil was used in full strength.

DR. SIDNEY OECHO asked if the oil remains in the larynx.

DR. BURNS stated that he had never seen a case of advanced tuberculosis of the larynx where the larynx became healed as in this case. His experience was that chaulmoogra oil seemed to relieve dysphagia very much in cases of ulceration, thereby enabling the patient to eat, but after treating about eighty cases he had never seen one heal as this one had. He thought it was indeed, most unusual.

DR. WM. MOORE said he thought Dr. Lukens had procured a remarkable result. Personally he found that chaulmoogra oil would do no good except for the time being. It helps swallowing, but does not cure. He thought it had more value where the lungs were quiescent.

DR. LUKENS (in closing) said that Doctors Jackson and Corman had examined his patient and the diagnosis had been tuberculosis. If he developed tuberculosis of the larynx, chaulmoogra oil would be his choice for treatment. He thought, however, that there are cases where the cautery should be used, and at times other things, but in the majority of cases chaulmoogra oil gives the best results. The oil was used in full strength, without preliminary injection of cocain, and after two or three treatments gave relief in swallowing. One-third of the oil remains on the larynx and two-thirds on the trachea. As chaulmoogra oil degenerates it should be comparatively fresh. In cold weather it should be slightly warmed before using.

Case Presentation: "Herpes Zoster in the Distribution of the Inferior Maxillary Division of the Fifth Nerve, Associated with Paralysis of the Seventh Nerve. Dr. James Hendrie Lloyd. (By Invitation.)

Dr. Lloyd said this disease is very interesting to the dermatologist although it is not a skin disease. It is a nervous disease. It is also interesting to the otologist, the laryngologist, the ophthalmologist and the physiologist. If it gets into the eye-ball it becomes very serious indeed, as it destroys the sight entirely. One of the characteristics of herpetic disease located in the ganglion is paralysis of the seventh nerve. The seventh and eighth nerves are closely connected, and for that reason there is often much auditory reaction, deafness, and so forth. Paralysis of the nerves in herpes is observed in other parts of the body. It has been seen in the muscles of the eye-ball and other cases have been reported in which the pharyngeal nerves were involved. There may be a number of these ganglia together. Dr. Lloyd referred to one case, that of a man who had been deaf since childhood in one ear. It seemed to have been an old, chronic, dry, middle-ear disease. When he was about thirty years of age he began to have pain in the lower part of his face, also vertigo and nausea. In a very short time a hereptic eruption appeared. It was in the area supplied by the third division of the fifth nerve. Very shortly after the appearance of the eruption he became completely paralyzed in the seventh nerve. Why did this happen? The area of distribution of the eruption was on the face. Getting up on the temple. It was not in the area described by Hunt, but at the same time it was associated with paralysis of the seventh nerve. Now the question remains why there should be this particular distribution of the herpes? Some have expressed the opinion that in this case more than one ganglion was involved. The distribution was not confined to one nerve trunk. He thought the matter was of importance. The distribution of the eruption was on the face, as well as on the fauces. Whether there was any on the back of the throat he did not know.

DISCUSSION.

DR. GEORGE W. MACKENZIE said he would like to report the first case of the kind he saw which was a number of years ago. It was a very interesting case from many angles. The patient came into the clinic with marked dizziness and herpetic blebs, which seemed to be different from the ordinary type they had been accustomed to seeing. This patient was a male, about thirty years of age. Over in the eye clinic he saw a girl. She spoke to him and said, "You don't remember me." I examined the patient and found she had hereditary syphilis. When you have blebs you should associate them with vertigo.

DR. LLOYD (in closing) remarked that a well known neurologist has written a treatise showing the distribution of the nerves of the face and that each side was totally different.

Report of Case: Brain Abscess Found During Operation. Exhibition of Brain. Dr. M. A. Schurter. (By Invitation.)

(To appear in subsequent issue of THE LARYNGOSCOPE.*)*

1. Laryngostomy for Cure of Laryngeal Stenosis. Presentation of Two Cases... 2. Post Typhoidal Cicatricial Stenosis of the Esophagus. Presentation of Two Patients. Dr. Louis H. Clerf.

Dr. Clerf said laryngeal stenosis is unusually interesting to laryngologists. It takes so many forms. It is seen in tuberculosis and acute infectious diseases. It was quite prevalent during the last epidemic of influenza, whooping cough, measles and so forth. The first case he showed was a post-diphtheria. The young man was admitted to the hospital in 1921. He had had diphtheria when five years old. Owing to his inability to keep a tube in his throat a tracheotomy could not be done, and he was returned home. Later he was brought up to see Dr. R. H. Skillern, who unquestionably advised a laryngotomy which the family refused. He also saw Dr. Jackson, who advised a laryngostomy. However, nothing was done for several months, when a laryngostomy was performed. A tube was worn and he was practically cured. At present he breathes through the mouth. He is now active in athletics and lives a normal life. He has a buccal voice, which is very good, considering he has not been using it much.

The second patient presented was a young man twenty-two years old, who was in the navy. In January, 1921, he developed an infection, involving the mucous membrane of the mouth, tonsils, pillars and pharynx. The same year he developed tenderness about the neck and hoarseness, which became worse. A tracheotomy was done. In the meantime, a leading laryngologist in Washington was called in consultation. He advised corking, hoping in that way to retain the use of the larynx. The patient finally came to the Bronchoscopic Clinic, and in August, 1922, a laryngostomy was performed. In September, of this year, they did a plastic operation. Since that time the patient's neck had been closed. One curious thing about the case was when the tracheotomy tube was taken out the patient could not get warm. Dr. Clerf called attention to one interesting thing in cases of stenosis of the larynx; during the acute stages it is not uncommon for the patients to have pain in the ear, which they complain of bitterly.

Report of a Case of Chronic Suppurative Otitis Media with Acute Suppurative Leptomeningitis and Brain Abscess. Dr. M. A. Schurter. (By Invitation.)

Mrs. D., age 28, colored, was admitted to the hospital on November 2. Her family history was negative. The patient had had measles and scarlet fever when a child.

1. Examination: On admission the patient was suffering with a severe and persistent headache without remission. She had pain and stiffness of the neck and was unable to push the chin to the chest. There was marked sensitiveness to light. For the most part the patient was

delirious and irrational. There was a constant cry and a condition of restlessness. Whenever an attempt to sit up was made there was a tendency to fall to the right. Jacksonian convulsions were present. It was observed that the left ear had a very foul smelling discharge which had become very profuse during the last two weeks of illness. It was then learned that this suppuration had been going on for a number of years. (Seven years.)

An attempt to X-ray the patient failed on account of the extreme restlessness. The right ear was douched and a very feeble response was observed after a considerable delay. There were no spontaneous ocular phenomena present. A spinal puncture was omitted on account of the suspected localized meningitis.

2. The urinary examination showed: Sp. Gr. 1022; a trace of albumen; hyalin casts; occasional squamous epithelium and a few leukocytes.

3. The blood picture was one denoting infection. Red cells 4,500,000; leukocytes 31,000; polymorphonuclears 81 per cent.

4. The patient was operated on Nov. 3, by Dr. Ursner. Removal of the cortex showed that the lateral sinus was very superficial and right close to the posterior canal wall. Suspecting a brain abscess the operation proceeded to the zygomatic ridge and squamous portion of the temporal bone. Working downward and forward in this region, to avoid the lateral sinus, an opening through the inner plate allowed the escape of about 2 dr. of pus which was under pressure. At this juncture the lateral sinus was injured but bleeding was controlled. Following this, communication with the antrum was established. After placing a drain into the opening made, the operation was terminated as the patient's condition was poor.

5. On Nov. 6, the patient died and having secured consent for autopsy the brain was removed. Dr. Case's report of the findings are as follows: The skull was of normal thickness. The lateral sinus was empty and torn as a result of injury. The petrous portion of the temporal bone was neurotic. On removal of the brain there was a considerable amount of foul smelling pus at the base which extended to the pituitary body and up laterally on the cerebrum. The left temporal lobe was the seat of an abscess 2 cm. in diameter which communicated with pus about the base of the brain. The anterior and lateral portions of the cerebellum in contact with the temporal lobe presented a superficial abscess. The vertex was free from disease. The cut sections of the brain do not reveal any involvement. The pus extended all the way from the cerebellum to the frontal lobe and was located between the dura and arachnoid. The drain which was placed at the operation was found in the abscess cavity of the temporal lobe.

Chronic Cicatricial Laryngeal Stenosis Cured by Laryngostomy. Dr. Louis H. Clerf, Phila.

Two cases of laryngeal stenosis cured by laryngostomy were presented. W. U., a young man, age 22 years, developed a pseudo membranous infection of the mouth and throat during January, 1921. The odium albicans and the spirillium and fusiform bacillus of Vincent were found. After six weeks this cleared up but was followed by a bronchitis, later an arthritis and during March, 1921, a laryngitis. Hoarseness increased, dyspnea developed and the neck became quite tender. Because of the progressive dyspnea tracheotomy was done April 3, 1921.

A diagnosis of laryngeal perichondritis with suppuration was made. Large quantities of pus were expectorated for about seven weeks. After the inflammation subsided and the laryngeal airway again restored, decannulation was attempted using partial corking. This seemed impossible as the stenosis of the larynx seemed to be increasing because of the secondary contraction of the scar tissue.

Laryngostomy was done August 23, 1922. Laryngeal packs were used until the larynx became epithelialized when the Jackson laryngostomy tube and rubber dilating apparatus was inserted. One December 19, 1922, the larynx seemed to possess a normal lumen and all apparatus was

removed. He was given a prolonged test period and on September 28, 1923, Dr. C. F. Nassau closed the fistula in the neck using a flap method. The patient is quite comfortable and has developed a very good voice. Phonation is carried on very largely by vibration of a mucosal fold on the right arytenoid.

The second case, W. M., a boy of 16 years, had diphtheria at the age of 5 years. He was intubated and wore the tube for three months, when a tracheotomy was done because of the lateral stenosis. He left the hospital one month later wearing the tracheotomy cannula.

Nothing was done until 1921, when he was sent to the Bronchoscopic Clinic. The larynx was found completely closed. The patient had developed a beautiful buccal voice.

Laryngostomy was done on July 27, 1921, and all dilating with the laryngostomy apparatus was discontinued during April, 1922. He has developed a good voice although he still likes to use the buccal voice. The laryngeal lumen is ample and the opening in the neck is now ready for closure.

January 8, 1924.

Remote Conditions Caused by Local Infection—from Ophthalmological Standpoint.* Dr. H. Maxwell Langdon, Philadelphia, Pa.

The subject assigned to me in this symposium, namely, "The Relation of Focal Infection in the Production of Ocular Disease", is so large that, to cover it completely would consume more time than is proper and I have therefore taken the liberty of not giving full consideration to foci which do not directly invite the interest of this society, as for instance, the appendix, prostrate, gall bladder and teeth.

That ocular disease does frequently result from a pocket of infectious material is a fact only recently realized as far as the medical professions as a whole is concerned. It is true that in some few cases of long ago casual references are made concerning the possibility of this or that condition resulting from bad tonsils or teeth but neither was the frequency of such condition grasped nor was the great variety of possible starting points realized. As an instance of this, iritis and uveitis, it may be remembered, were ascribed to syphilis, tuberculosis, gout, rheumatism, diabetes or gonorrhoea, and yet in all probability one-half of the cases of these combined conditions are the result of focal infection.

To be sure all cases of disease which occur in an individual in whom a pocket of infectious material is discovered may not be the results of this infection any more than all conditions arising in patients with a positive Wassermann are due to syphilis, and it is very easy to reach a wrong conclusion as to the relation between, for instance, a case of iritis and infected tonsils unless a most complete study is made of the patient. Only the utmost care will keep one from being misled. Ocular disease arising from infectious foci is variable in type, similar conditions resulting from quite different starting points, for instance, iritis may result from sphenoidal infection, tooth root abscesses, tonsillar involvement or appendicitis—and infection in the same locality may produce widely differing results in different individuals, sphenoidal disease causing retro-bulbar neuritis in one case and a violent iritis in another. In the experience of the writer it is impossible in the majority of cases, to feel sure that the originating site has been discovered unless a most complete examination of the patient has been made. Ocular conditions which have been reported as due to focal infection arising from various sources include—retro-bulbar neuritis, neuro-retinitis, retino-choroiditis, iritis, interstitial keratitis, episcleritis, and blepharitis, and the sources included by various writers are the various nasal accessory sinuses, tonsils, teeth, appendix, gall bladder and prostate.

The ocular conditions being the predominant one, these cases are usually first seen by the ophthalmologist and the first responsibility is his. In some instances one can feel fairly certain of the starting point of the

trouble, but these cases are few, by far the greater number being quite obscure and in many, exploratory procedures alone settling the question. In one case of the writer's, three rhinologists saw the patient, all agreeing that the supposition that a sinusitis was the cause of the violent kerato-irido-neuro-retinitis was wrong and that the badly infected tonsils were causing the ocular disturbance, an opinion which seemed proven when there was great improvement in the eye following an expression of infectious material from the tonsils. Theye were removed but the eye grew steadily worse until vision was reduced to perception of concentrated light, where it remained for three days until, at the writer's request, sphenoidal drainage on the affected side was established and in less than a month vision was normal. The writer knows of a case treated by an acquaintance in a neighboring city where total blindness resulted, both eyes being affected. All other possible causes had been excluded but the rhinologists were so sure the sinuses were healthy that they refused to operate and though the ophthalmologist felt the cause was in either the sphenoids or ethmoids he did not insist on action.

In seeking the cause of a case of retro-bulbar neuritis with central scotoma it must not be forgotten that it is a common result of tobacco-alcohol toxemia, at times a part of disseminated sclerosis and that it also occurs in tabes; and that all these must be considered and eliminated, but at the same time it must be remembered that the patient may have sphenoidal disease even though he has been a heavy consumer of tobacco and ethyl alcohol, or may have some tremors or a slurring speech, or lost knee jerks and a positive Wassermann. Iritis, likewise frequently results from syphilis or tuberculosis, but subjects with these infections may also have had tonsils or teeth with root abscesses.

Cases do occur which seem to point directly to a focus of infection as a starting point but which in reality are from a systemic condition. Recently a colored woman, aged 33, reported at the dispensary of the Presbyterian Hospital complaining of blurred vision of the right eye, accompanied by some headaches. Examination showed no external changes, vision of O.D. 6/15, O.S. 6/5. O.D. disc hyperemic, with blurred margins, and swollen about two diopeters above the fundus level, with some fullness of the veins but no other changes. The field showed enlargement of the blind spot, but was otherwise normal. The left eye was normal in all respects. Rhinological and X-ray examinations showed the sinuses negative, though it seemed that the most likely cause was either sphenoidal or ethmoidal involvement. Physical examination showed an unusually high blood pressure and urine filled with albumin, the condition being a unilateral neuro-retinitis from nephritis. And yet, this case is not so very different from one of a unilateral neuro-retinitis from tonsillar infection which was operated on by Dr. Robert Hunter at the Presbyterian Hospital:

Miss E. B. V., age 17, was first seen on October 27, 1923, with blurred vision of O.D., which had then lasted for two weeks. No other symptoms. O.D.V. was 5/35 with a large central scotoma, the disc was hyperemic with some blurring of the temporal margin. O.S.V. 5/5, the eye being normal. Wassermann and Von Pirquet tests negative, sinuses, blood, urine and teeth negative, but the tonsils were badly infected, and were immediately removed. On November 4, O.D.V. was 5/15, the disc still being swollen with a complete muscular wheel. By November 24, O.D.V. was 5/5 and the fundus was normal except for slight hyperemia of the disc and one or two "spokes" remaining of the macular change. Central color vision was normal.

In both these cases we have a toxemia, in the one from the kidneys and in the other from the tonsils and in each we have an inflammatory reaction in only one eye, whereas it would seem reasonable to expect a binocular involvement.

Interstitial keratitis in by far the greater number of cases results from hereditary syphilis, and is usually bilateral. Within the past two years the writer has seen three unilateral cases, not typical however, as they

lacked the corneal vascularity of the usual case, all from nasal conditions, one with atrophic rhinitis which was treated by Dr. Ralph Butler and made a good recovery, vision returning to 5/8 from 1/60, one treated by Dr. James Babbitt, vision returning to normal from 5/25, and the third treated by Dr. Seth Brumm, vision of the affected eye here falling to 5/30, but now being 5/5 minus. The last mentioned, a lad of twelve, also had badly infected tonsils, and he was the only one who had much pericorneal injection, there appearing at the upper limbus a deeply engorged area resembling the well known "salmon patch" of the specific variety.

In the writer's experience ocular disease resulting from any site of focal infection is usually unilateral but the following case will show that a bilateral condition may result:

Miss E. B. S., age 21, was seen on March 4, 1922, complaining of blurred vision in each eye. Examination showed no changes in any structure of either eye. O.D.V. was 5/10, the best corrected vision being 5/9 plus; O.S.V. 5/8 partly, capable of correction to 5/6. Examination of the visual fields showed normal peripheral limits with a relative central color scotoma, the diagnosis being a retro-bulbar neuritis. Physical examination showed a rather low blood pressure, 11-65, hemoglobin 68 per cent, reds 3,250,000 and whites 5,000. Wassermann weakly positive, but no other findings. Examination of the teeth showed them in good condition. The tonsils had been removed and the sinuses were reported as healthy. Vision not improving under iodids, another rhinologist, Dr. Ross Hall Skillern, was asked to see the patient, an he felt that there was enough sphenoidal infection to account for the trouble. The sphenoids were drained and in six weeks vision was 5/5 plus in each eye.

It was believed for a while that this case was an early manifestation of involvement of the central nervous system but the ocular improvement after operation, which is still maintained, visual acuity being 5/5 plus three weeks ago, and the continued absence of the other signs makes it seem positive that it was a local condition.

At times various ocular manifestations result from an infected sinus, different ocular structures suffering from the infectious material at different times, a state of affairs which the following case exemplifies:

Mr. B. H., age 25, first seen March 13, 1917, with a history that O.D. had been inflamed four years previously, with some recurrence at times. O.S. had been sore the previous week. O.D. was normal in all respects, with a vision of 6/5. O.S. showed intense ciliary injection, with a cloudy cornea, the pupil filled with exudate and a slight hypopyn; vision being perception of hand motions at one foot. The eye had been under indifferent treatment. Under suitable local and general measures, conditions improved so that in four days the exudate in the pupil was absorbed and vision was 6/25. The Wassermann was negative, the teeth were in good condition but the sphenoids were infected. On April 27, 1917, a hemorrhagic retinitis developed in O.S. which treatment quieted, the nasal treatment consisting in the use of the suction apparatus. In June, the right eye became inflamed, but the attack was light. In August, 1918, O.S. was again involved, the iris being so congested that there were hemorrhages from it into the anterior chamber, leeches being required to lessen the iritis hyperemia; a similar attack occurred three months later. In December, 1919, a retro-bulbar neuritis with central scotoma and some edema of the disc appeared in O.D., vision falling to 6/30, to return to 6/5. Radical operation has been refused, though he has had several ocular inflammatory attacks, each aborted by nasal treatment.

This case also illustrates a point which has been impressed on the writer more than once and that is the apparent futility of the suction treatment in these conditions—he has yet to see a case cured by it, a fact also shown by the following case:

Mrs. E. T. L., age 60, reported on July 19, 1920, with a history that O.S. had been sore since the preceding day. Examination showed O.D. normal with a vision of 6/10. O.S. slight ciliary injection with two posterior synechia and a vision of 6/15. Examination showed normal

urine, negative Wassermann, two teeth with apical abscesses and an involvement of the ethmoid cells. The teeth were extracted and the ethmoids treated by suction, the patient however, not objecting to more radical procedures. This attack cleared up, but two months later O.D. was involved. In January, 1921, the left eye was again involved, which lasted with short intermissions, until August of that year, when the left antrum and ethmoid were cleared out, with an almost immediate improvement in the ocular condition, and without return up to the present. Corrected vision is normal, and except for a few pigment spots on the anterior capsule of the lens, the eye is undamaged. One of the interesting things about this case was a sensitization to atropin after a long period of use and the necessity of using scopolamine; also interesting was the long dilation of the pupil without increase in ocular tension in a woman of sixty.

In some instances even after operative procedures we have recurrences, drainage either ceasing, or a hyperplastic cell lining becoming again virulent, as is shown by the case of:

Mrs. A. H. C., age 30, seen first February 15, 1915, for refraction. On December 7, 1915, she again reported this time with an inflamed right eye, which went on to a severe iridocyclitis with much K.P. and iris engorgement. Physical examination including Wassermann was negative. but nasal examination showed a septum deflected to the right with acute infection of the ethmoid and sphenoid cells. They were treated, and by December 27, 1915, the eye was white. On August 1, 1919, there was another outbreak in the same eye, being clear in two weeks. A third attack started December 19, 1922, with much congestion of the iris and chemosis of the conjunctiva. Pus was obtained on opening the ethmoids, and by January 2, the eye was again almost white, with normal vision.

Another type of return of the ocular disease after a complete recovery is shown by the case of:

Mrs. C.C., age 33, first seen on February 3, 1923, with a right eye which had been sore for about twelve hours. There was considerable corneal haze, and ciliary and iritic congestion, the vision being 5/10. O.S. was quiet with a vision of 5/5. Teeth, Wassermann and general physical examination were negative. She was seen by three rhinolaryngologists, all of whom said the sinuses were negative but the tonsils were badly infected. Following an expression of infectious material from the tonsils the eye greatly improved, seeming positively to point to the source of infection. The tonsils were removed February 9, 1923, and the eye became worse, the pupils filled with exudate, cornea densely hazed and the conjunctiva so greatly chemosed that it overhung the limbus; vision at this time was scant light perception. In addition to the other local remedies, leeches were applied to the temples with no great relief.

On February 14, there was a small hypopyon. Consultation was again held with the laryngologist. Dr. Ross Hall Skillern, who could still find nothing to suggest sinus involvement. The eye continued in its severely inflamed state, and on February 18, the writer insisted on amputation of the right midde turbinate, which was done by Dr. Skilern the following day, when there was found in the sphenoid a thin blood tinged serous fluid. In forty-eight hours the improvement in the eye was most remarkable, the cornea being clear enough to see that the vitreous was quite hazy. In three days more the vitreous was clear enough to see that the disc and retina were quite edematous. Improvement, however, continued, and in four weeks from the nasal operation corrected vision was normal.

The eyes remained in good condition until the following September when O.D. became again involved, by a moderately severe iritis. Dr. Skillern found the opening into the sphenoid still well open, but that there was involvement of the ethmoid; on the establishment of drainage of this cell the eye promptly improved and in two weeks conditions were normal.

Here in all probability there was an infection of the ethmoid at the time of the first attack but of such low virulency as to be harmless, later becoming active.

Dr. Durrick Vail of Cincinnati has reported three cases of ocular disease resulting from sinus involvement where all the nasal findings including Roentgenological examination were negative. This condition is possibly more frequent than we realize and its possibility should be born in mind. Such a case is that of:

Mrs. M. B. W., age 52, seen on January 23, 1917, complaining of blurred vision of O.D. with pain in the right frontal region following influenza. O.D.V. was limited to counting fingers at 2 feet, the direct pupil response was abolished but consensual reaction was good, a condition which was reversed in the left eye. The right disc was hyperemic with blurring of the margins. The left eye had normal vision, full field and no changes except the loss of consensual pupil activity. Dr. George B. Wood could find no signs of sinus involvement, and X-ray examination by Dr. H. K. Pancoast was negative, but so plain did the underlying cause seem to be that the sphenoids were opened on the writer's insistence on January 28, 1917. Almost at once an improvement in vision was noticed, and by February 2, 1917, vision of O.D. was 6/20, and one week later corrected vision of this eye was 6/6.

It has been suggested that this case was functional rather than organic, but the alteration of pupil activity and the changes in the optic disc leaves no doubt in the mind of the writer that it was a case of actual disease rather than one of hysteria.

Our knowledge of the method of production of these end results is in some instances not conclusive. In cases where the site of origin is in the appendix or tonsils it would seem that the blood stream is the path of infection, where we find an acute ocular inflammation from either teeth or sinuses either the blood or lymph channels may be the routes. In cases of retro-bulbar neuritis with central scotoma there is a distinct divergence of opinion as to whether it is the result of inflammatory reaction in the trunk of the optic nerve resulting from infectious material transcending from the involved sinus or whether it is from mechanical pressure on the nerve by the thickened sinus wall. It is a recognized fact that the papillo-macular bundle of the optic nerve is very susceptible to toxic conditions much more so than is the remainder of the nerve trunk. The proponents of the pressure theory feel that after the sinus is opened the disappearance of the scotoma is too rapid to have been the result of an actual inflammatory condition, but it must be remembered that in tobacco-alcohol amblyopia, an identical condition save for the toxic agent, the return of visual activity is in some instances very rapid after the cessation of the use of the irritating substances.

The writer feels that in these cases there is in all probability an actual inflammation of the nerve, and that if pressure caused the visual disturbance, in some cases there would be a contraction of the peripheral portion of the visual field rather than a central scotoma.

Periapical Infections from the Exodontist's Viewpoint.* Dr. Earle C. Rice.

One of the most serious problems that confronts the dental profession today is periapical infection—focal infection.

Few men face pulp extirpation without grave misgivings that exodonist may need to be called upon eventually to remove the infection, which is so often the sequel of the most careful and painstaking technique in root-canal work.

No thinking dentist will look lightly upon the presence of pathogenic organisms that have found their way into the periapical tissues.

It must be borne in mind that the anatomical construction of a tooth defies the removal of the dental pulp in its entirety: for the odontoblasts and their protoplasmic prolongations, that terminate in the stratum granulosum, must remain as non-vital, organic tissue inviting the growth of pathogenic flora which in time gain access to the periapical tissue.

The internist has sounded the warning, and are confronted with the serious disorders directly traceable to focal infection.

It is the purpose of this paper to touch briefly upon the findings of your essayist in collaboration with the internist, the bacteriologist, and the roentgenologist. Innumerable cases of pericarditis, endocarditis, micarditis, cholecystitis, arthritis, myositis, neuritis, choroiditis, conjunctivitis have been directly traced to dental focal infections, and complete recovery noted upon the removal of the teeth so effected.

It has been further noted, that many teeth with non-vital pulps roentgenographically negative, when extracted and placed in the hands of the bacteriologist, reveal the presence of virulent organisms.

It is a notable fact that the roentgenogram is only a record of relative densities of tissues photographed, and the diseased processes that do not effect a destruction of tissue, and hence their density, are not shown. It is therefore a matter of co-operation between the roentgenologist, the internist and the exodontist to determine the fate of the negative tooth, and the office of the bacteriologist to prove their findings.

It is a lamentable fact that many dentists preserve *in situ* teeth that show unmistakably in the roentgenographic records that the periapical tissues are affected. It can not be too strongly urged that all teeth showing these radiolucent areas should be condemned to the forceps, and also many that are thought to be negative.

Your essayist holds that the exodontist, by virtue of his association with the bacteriologist and his examination of teeth and sockets after extraction, is in a better position to pass judgment upon a suspect, than any other specialist. He has viewed many thousands of cases before and after, and is less liable to err on judgment than those who attempt to save pulpless teeth.

The following technique has been employed by your essayist in making cultures of infected teeth: The bacteriologist supplies a Petrie dish, containing a film of blood agar, and a number of sterile tubes.

The suspected teeth are carefully removed avoiding contact with the oral tissues. The root apices are clipped off with sterile excising forceps, placed in the sterile tubes and numbered.

The curettage from each socket is planted in the blood agar and numbered to correspond with the tooth removed from the socket. These exhibits are then sent to the bacteriological laboratory, where the flora are incubated.

Your essayist is debtor to Dr. Russel Richardson for this step in the technique, and also for his deft laboratory methods which develop the last word in the diagnosis.

After more than thirty years of earnest investigation, your essayist has reached the seemingly radical conclusion, that the death of the dental pulp from whatever cause, is an invitation for the exodontist to test his skill.

Sooner or later all non-vital teeth will show infection, and the blood of the victim is on the hands of him who fails to remove it.

The following case report is significant: A young physician, thirty-five years of age, suffering from cholecystitis, presented himself for the removal of three teeth each showing granuloma in the roentgenogram. The bacteriological findings were: Streptococcus hemalyticus. He continued to suffer. The internist advised the removal of four other non-vital teeth roentgenographically negative. This was done and streptococcus viridans were found. His recovery was prompt and complete.

It will be noted here that the seemingly innocent teeth were the culprits.

The exodontist's viewpoint is influenced by the opinions of hundreds of practitioners. He sees their successes and their failures. He acts upon their judgment, tempered by his own. His close contact with the findings of collateral workers brings him in closer touch with reality in disease

processes that are of dental origin. He does not urge extraction except as a means to an end: primarily to remove infections that militate against the well-being of mankind.

January 8, 1924.

DISCUSSION.

DR. RUSSELL RICHARDSON made a plea for closer co-operation in the work that the laryngologist and the laboratory man do. All of them had been familiar in the hospitals with the old-fashioned way of the surgeon when opening abscesses, putting the swabs of cotton aside and sending for the laboratory man when he had time. That method would do well enough in some cases, but other more or less close methods of handling material for culture would also work well. He said one could not do good work with bacteriological cultures unless there was close co-operation between the doctor and the laboratory man who helped him. Personal co-operation. They must get together with the methods they were going to use, each bringing his knowledge so that there would be no chance for a hitch. Unfortunate things result if the work is careless. Many of the organisms with which they deal in the regions of the naso-pharynx, tonsils and so on are not easy to grow. They will not grow if not looked after. In some of the cases that Dr. Langdon mentioned, Dr. Richardson though that, perhaps, if there had been co-operation between a nose and throat man and a laboratory man, who would go to the hospital with his outfit and examine the portion of the nasal passages that looked suspicious to him, that it might have been possible to find the site of the organisms. In regard to taking a swab of cotton and pushing it against the tonsils, one gets some material, but it will give very little results of any moment. The organisms may dry. You have mixed organisms from the crypts of the tonsils and those on the surface, but you will find few pathogenic organisms. However, if one got down into the tonsils and down in one-fourth to three-fourths or one-half inch of the crypt, a real bit of the material that tells what is at the bottom of the crypt could be obtained. A large number of people carry some streptococci in the throat and many pathogenic organisms of other sorts. He thought more attention should be paid to the number of organisms, as well as to the variety.

DR. G. W. MACKENZIE thanked those taking part in the program and said the symposium was the best that the Society had ever been treated to. It was pleasing to see how progressive the men on the program were from one end to the other. One case that Dr. Langdon spoke of was that of recurring attacks of neuritis. Dr. Mackenzie happened to have known of a case of neuritis of one eye. Careful search of the sinuses, blood, urine, stool and so forth, also complete blood examination, did not reveal the cause of the neuritis. Several months afterward the patient reported again for the same thing. He was for the second time searched very carefully and nothing found but an impacted tooth and tubular swelling on the right side of the throat. The tonsils were removed and the eye condition of the patient improved apparently over night. In about nine months the patient came back again with a recurrence. He went to Atlantic City and while there developed a severe cough and subsequently had an operation for cholecystitis with gallstones. It was reasoned that in this case the primary infection was in the tonsils, but they were removed too late. Dr. Rice's paper impressed him when he stated that it is practically impossible to sterilize the pulp chamber of the tooth. Speaking of non-vital teeth, it is possible that one may take a three-pronged tooth, destroy it, and remove it intact. In regard to the extraction of teeth, he thought they all agreed that it is a surgical operation. In cases where there is pus they were making a plea for curettment.

DR. SAMUEL SKILLERN wished to second Dr. MacKenzie's statement that this was the best symposium the Society had ever had. He enjoyed all the papers, particularly Dr. Daland's. Fourteen years ago he listened to Dr. Daland make some of the same statements in regard to focal infection. At that time they all thought he was focal infection mad, and had many discussions as to what a drop of pus would do. Dr. Skillern read a paper several months ago in which he stated that in his opinion 90 per cent of all tonsils were infected, and Dr. Daland's statement had brought home to him that preventative measures in the removal of tonsils is an excellent thing. He declared that he intended to remove his baby's tonsils as soon as she is old enough, in order to prevent future trouble.

DR. BENJAMIN H. SHUSTER said he was impressed especially by a point brought out by Dr. Langdon when he said in severe eye conditions consult the rhinologist as well as the ophthalmologist. He spoke of one of his own patients who had been up every night with palpitation of the heart. Removal of the tonsils was suggested. The man hesitated but finally consented. Immediate relief followed the operation.

DR. MEYER SOLIS COHEN thought they were accustomed to associating focal infection with diseased tissue but did not always realize what causes the infection. When the patient's resistance has been increased by means of vaccine, infection in other parts of the body disappears. After the nose and throat are carefully examined and nothing is found, that does not mean there is no infection there. There may be, although the eye can see no evidence of the disease. Everyone has organisms of some sort in the upper respiratory tract. When you remove the tonsils, for instance, you remove the organisms in the tonsils, and no other. The organisms in the nares remain there. They have many cases where the focus of infection is in the tonsils and the patient is not cured by the removal of the tonsils. One can easily see that if organisms are present in the rhino-pharynx they stay there and grow. The virulent ones the patient will carry in his blood, and that is where the secondary focus is not removed. Organisms in the gall-bladder remain there. Only when a vaccine made from the secondary focus of infection has been given can the primary infection be cured.

DR. MAXWELL LANGDON (in closing) said in reference to the case he referred to that the patient's doctor was particularly well acquainted in New York. He had three of the best laryngologists in that city but the patient went totally blind in each eye, for although there was a nasal condition, he could not get any of these men to operate because they were afraid they would do more harm than good, and be blamed. He himself knew too little about intranasal operations to speak with authority but from what he had seen done in Philadelphia, he thought that much harm could have been saved had those doctors had a little more firmness of their convictions.

DR. R. H. SKILLERN (in closing) said he thought when there was any question whether the tonsils were diseased or not the best thing to do was to send the patient to a competent bacteriologist. Have him get down to the bottom of the deepest crypt he can find and make him a culture from it. He thought vaccine should be made from culture made deep. He did not place much reliance upon the bacteriological finding unless one of the varieties of streptococci were present. That is either viridens or hemolyticus. Vaccine from these is nearly always followed by favorable results.

DR. EARL C. RICE (in closing) wished to lay stress on the treatment of the socket after removal of the tooth. If there is a condition of granuloma it should be traced out without injury to the granuloma.

DR. JUDSON DALAND (in closing) wished to emphasize that the absence of pus does not necessarily mean an absence of focal infection.

February 5, 1924.

Occipital Abscess in Mastoid Disease. Presentation of Patient. Dr. Henry Dintenfass.

(To appear in subsequent issue of THE LARYNGOSCOPE.*)*

Acute Mastoiditis, Apparently Primary, in an Infant Seven Months Old. Dr. Arthur J. Wagers.

(To appear in subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. HENRY S. WIDENER said that he had had the pleasure of watching Dr. Dintenfass' case both before and after the operation, and that there were many interesting things about it. The first point of opening was back beneath the mastoid. They could find no connection with the mastoid. This wound healed very nicely but the occipital wound failed to heal, and there was a discharge from it which was almost exactly like a tuberculous pus (creamy white). In fact, the whole aspect of the case was that of tuberculosis but the wound was examined, and they were unable to prove it.

DR. GEORGE W. MACKENZIE remarked that the bacteriological findings as they get them are not always absolute.

DR. WILLIAM SHEMELEY said he enjoyed both papers very much. He took the liberty of examining the mouth of Dr. Dintenfass' patient, and found much evidence of pyorrhea. It seemed to him that that should have been gone into. His second thought in regard to this patient was whether a Wassermann had been taken. Dr. Wagers' case was particularly interesting to him because it reminded him of the first mastoid case that he had the privilege of having. It presented many of the same characteristics. In his case, the whole temporal bone was necrosed. He wished to know if Dr. Wagers had had a culture made from the pus.

DR. DINTENFASS (in closing) said a Wassermann had been taken, and the result was negative. The patient's teeth had also been looked into, but she refused to have anything radical done to them.

DR. WAGERS (in closing) stated that he did not have a culture of the pus, as the child was first seen by him at nine in the evening, and after examination was operated upon at once. At that hour the laboratory was closed.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

December 14, 1923.

The Relation Between the Vestibular Apparatus and the Cerebellum, from an Evolutionary Standpoint. Dr. Wells P. Eagleton.

Personal Observations and Critique of the Diagnostic Significance of the Labyrinthine Reactions. Dr. S. J. Kopetsky.

(Published in THE LARYNGOSCOPE, *April, 1924.)*

DISCUSSION.

DR. LEWIS FISHER (Philadelphia) said that after the somewhat gloomy impression conveyed by Dr. Kopetsky's observations on the examination of the labyrinth, he would try to say a few cheering words about it. There was a time when everything was claimed for neuro-otologic examinations; every writer on the subject claimed wonders for it. The pendulum has now swung the other way and we frequently near how little can be done with it or how useless it is. It is true that the nerve pathways constituting the vestibular mechanism have not as yet been fully demonstrated, histologically. The difficulties underlying such a demonstration were pointed out by Dr. Eagleton. As a matter of fact, though, a goodly portion of the mechanism has been definitely worked out. By means of the Golgi preparations one can trace the fibers from the vestibule to the eighth nerve, the brain-stem and the cerebellar cortex.

In addition to this, we have clinical evidence which must not be disregarded. Every otologist has seen cases of chronic ear suppuration with a fistula in the external or horizontal semi-circular canal where the slightest pressure on the fistula produces instant movement of both eyes. This direct stimulation of the labyrinth always produces a definite eye-response. We also know that other types of labyrinthine stimulation produce similar definite eye-movements. Irrespective of histological demonstrations, common sense tells us that there must be a nerve connection between the ear and the eye muscles producing these movements. Clinical experience also teaches us that where there is a lesion between the ear and eye-muscles, such as is produced by a destruction of the laby-labyrinth, or by sections of the eighth nerve, the same ear stimulation will not produce these eye-movements. Why then not utilize these facts clinically in our study of cases with symptoms referrable to affections of the vestibular mechanism?

In going over the records of the past few years, I find 92 cases of brain lesion that have been studied by these tests, which were verified by operation or autopsy.

There were 20 cases of angle tumor where the vestibular diagnosis was verified; 2 cases where it was not verified.

21 cases of cerebellar disease proved the findings of vestibular examination at operation or death; 9 cases not proven.

18 cases of cerebral tumors examined were in accord with the vestibular findings; 4 cases were not verified.

5 cases of pituitary tumor verified the vestibular tests; 1 did not.

1 case was suggested as a lesion in the cerebellar peduncles and was verified by autopsy.

Of the cases that were not proved, the suggestions from the vestibular tests may or may not have been wrong. If the tumor is not located at operation it does not necessarily mean that it is not there. For instance, while in Detroit some years ago, I examined a case that I thought, from the vestibular tests, to be one of cerebello-pontile-angle tumor. I left

the following day. The patient was operated upon later, but the surgeon did not find a tumor. Three days later I had another letter stating that the patient died and an autopsy showed a large tumor in the angle.

Examination in all these cases was made from a purely otologic standpoint. Not being a neurologist, neurologic tests were not attempted. Most of the patients were sent down to the ear clinic without a chart or history. They were questioned as to the chief complaint and general medical history and were put through the complete routine vestibular examination, following the outline of the chart suggested by Dr. I. H. Jones and myself. From these findings a possible diagnosis was suggested —always suggested but never actually made—the thought being that it is not the business of the otologist to make neurologic diagnoses. That the suggestions in the 92 cases quoted should have been correct in such a large proportion of the cases, convinces me more than ever of the value of such an examination, especially since they were made by one not trained in neurology. I believe that it is convincing proof of the value of careful observation and recording of the clinical phenomena and responses obtained.

A study of what is actually going on within the labyrinth while it is being stimulated is, of course, most interesting, and any contribution towards it must be of great value in helping us understand the whys and wherefors of the responses obtained when patients are examined. However, a lack of a complete understanding at this time need not impair our faith in the clinical value of our observations on the results obtained. The various laboratory experiments have succeeded in creating a great deal of confusion; thus Dr. Kopetsky quotes the investigations of Bories, who found that destruction of the semi-circular canals in pigeons forever abolishes nystagmus after rotation. On the other hand, Maxwell reports that he succeeded in destroying the semi-circular can as in animals, but that subsequent rotation demonstrated that eye-movement could still be produced. Both men are brilliant observers but the decidedly opposite results obtained certainly doesn't help in finding out what is happening in the human labyrinth. What is true of the pigeon may not be true of selacians, and equally untrue of the human ear. We always thought that rotation with the head 30 degrees forward stimulated the end organs within the horizontal semi-circular canals and that douching with hot or cold water with the head 60 degrees back stimulated the same canal. According to the experience of assayist, apparently supported by the researches of others, we have all been wrong.

Let us for a moment analyze a few facts which we believe to be true. In the first place the end-organ of the vestibular mechanism is always a ciliated hair cell. If we trace its development from the very lowest forms of life, be it an open pit as it is in the very beginning, or a tube sunk below the surface of the skin as it occurs somewhat later, or whether it is a complicated system of canals with saccule, utricle and cochlea as seen in the highest forms of life, the real perceptive end-organ is always a ciliated hair cell. In the open pit with the animal living in water the gross wave movement of the water itself acts upon the ciliated hair cell. In the more complicated organ it is body movement of the animal that generates the wave for the stimulation of the ciliated hair cell, but it is always a wave movement of fluid. Thus in the cochlea it is the stapes and other bones that set the lymph fluid vibrating; in the labyrinthine fistula test mentioned before, manual pressure with a probe disturbs the lymph; we are justified in believing that in the rotation test we also stir up the labyrinthine fluid; the caloric test would certainly tend to show that we induce some sort of lymph movement. With the head in a given position we are able to reverse the direction of the eye-movements by changing the temperature of the water from cold to hot, or, what is even more convincing proof is elicited in the experiment where, without changing the temperature of the water, we are able to reverse (not change its character, but actually reverse) the direction of the eye-movements by simply moving the head through an arc of 180 degrees. To bring about such a reversal it would seem we must

have reversed the current, be it molecular or what-not, within the labyrinth. When an individual is rotated with the head 30 degrees forward, a certain definite portion of his labyrinth is being stimulated. This produces the movement of both eyes in a horizontal plane through innervation of the external and internal recti muscles. Now when the labyrinth is stimulated by another means, such as caloric test, and we produce the same eye-movements—that is, again innervation of the internal and external recti—it is fair to assume that the identical portion of the labyrinth was stimulated. In this way we have come to believe that turning the patient with the head 30 degrees forward stimulated the horizontal canal, while douching with hot and cold water with the head 60 degrees back also stimulated the same horizontal canal. In a similar manner we have come to believe that rotation with the head forward or backward stimulated the same portion of the labyrinth that the caloric test stimulates with the head in the so-called upright position. With these as a basis, phenomena obtained on the examination of a large number of individuals were observed and tabulated. Their application in the study of clinical cases produced results just cited in the 92 cases. Dr. Kopetsky is inclined to believe that our conception as to the portions of the labyrinth stimulated needs complete revision. With this I cannot fully agree in the absence of more convincing proof. From a clinical standpoint, what real difference does it make which particular crista is stimulated as long as experience tells us that when a certain test is performed in a certain way, well known and definite phenomena appear? Does it not remind one of the story of the discovery that the Iliad was not written by Homer at all, but by another man of the same name who happened to live about that time? In a word—it will be wise to hold on to the older thoughts since they have proved so useful before embracing the new. However, we all realize that many of the findings obtained in the examination of patients are very baffling and those quoted by Dr. Kopetsky represent real difficulties in the attempt at a thorough understanding of the workings of the vestibular mechanism. Any light thrown upon these questions will eventually be very helpful in making this type of examination even more useful than it has been to date, and his efforts in that direction deserve the highest praise.

I fully agree with Dr. Kopetsky that the neuro-otologic tests are absolutely indispensable in our routine ear work, and that their aid to the neurologist in brain tumor cases is simply incidental. There is a type of tumor, however, that is more in the realm of otology than in that of neurology. I refer to tumors of the cerebello-pontile-angle. The earliest symptoms in these cases are deafness, tinitis and vertigo. With this triad the patient usually consults the otologist first. As these lesions are slow-growing, it is not infrequent for the otologist to continue treating such a patient with tubal inflation, or perhaps carry out a tonsillectomy or some nasal surgery, only to discover later that his patient had a cerebello-pontile-angle tumor. Fortunately the neuro-otologic findings in a case of this type manifest themselves quite early and are very definite, so that if the otologist is at all alert, he will be able to make a diagnosis of such a tumor long before any neurologic signs make their appearance. Since angle tumors are relatively common, the otologist at once assumes an important role in the consideration of brain tumors in general, and his peculiar fitness to diagnose them early, throws the responsibility directly upon him.

In relation to other brain tumors his role is purely that of auxilliary. Of tumors located elsewhere than in the cerebello-pontile-angle, the neuro-otologic findings will assist most in cases where the cerebellum is involved. Frequently the neurologist finds considerable difficulty in differentiating between lesions of the cerebrum and cerebellum. If the otologist can only assure his neurologic confrere that the posterior fossa is normal he is most helpful. In studying for possible affections in the sub-tentorial region, the vertigo and so-called past-pointing tests are most valuable. Lesions affecting the cerebellum are very prone to interfere with the "motion-sensing" or so-called vertigo responses; that is, if a

normal individual is rotated, let us say to the right, 10 times in 10 seconds and the chair stopped he will feel himself revolving in the opposite direction for about 25 seconds. If the cerebellum is affected the duration of the vertigo is considerably reduced, instead of the 25 seconds it may last 3, 5 or 10 seconds. With this reduction in vertigo there will also be noted an absence or impairment of the past-pointing responses. In my experience this test is quite reliable, especially when the responses are present, by which I mean that any individual who exhibits more or less normal vertigo with good past-pointing with both arms in both directions can be considered as having no cerebellar lesion. An absence of these responses, while strongly suggesting, is not as good evidence as when these same responses are present.

In cerebral lesions a neuro-otologic examination might occasionally help to determine the side on which a lesion is located—this by observing carefully for the presence or absence or quality of the quick component of induced nystagmus. The exact origin of the quick component of nystagmus is still undetermined. From clinical evidence at hand we believe that it is cerebral in origin, somewhat subcordical, not quite high enough to be on a level with the centers of consciousness. In several cases it was shown that a superficial subcortical lesion appears to interfere definitely with the quick movement of both eyes in the direction opposite to the location of the lesion.

In conclusion, I would like to emphasize the thought that neuro-otology was never meant to supplant neurology. An individual with neurologic symptoms should be under the care of a neurologist, who shall advise the necessity of an otologic examination if, in his opinion, such an examination will help him in a diagnosis of his case.

DR. FREISNER said that there was such a formidabe array of neurological talent present that he preferred to defer to them rather than to speak at length in the discussion of this subject, but that he would like to hear more from Dr. Eagleton about the compensation which he had mentioned as one of the functions of the cerebellum. There is a curious thing with regard to the interaction of the internal ears; when they are both intact they are entirely unilateral organs, opposed more or less to one another. When one is destroyed, the remaining internal ear acts as a unilateral organ for a while until compensation takes place; it then assumes more or less a bilateral function. How much the cerebellum plays in this adjustment is questionable . . . we know for instance, that after the destruction of one labyrinth . . . if the hetero-lateral frontal lobe in the dog is destroyed so-called compensation takes place very slowly; after all, the compensation is perhaps more a cerebral function than a cerebellar.

Dr. Kopetsky's presentation was very interesting. No one knows much about the function of the otolithic organs; it is almost impossible to experiment solely on them. That brings to mind another fact: Many years ago otologists thought that where the labyrinth was destroyed by suppuration a fistula test was still positive, because it was found that by the introduction of a probe into the opening through the bony wall of the diseased labyrinth there was still some reaction.

Boris' contribution was interesting and valuable, but there is this much to be said about it: that where one destroys the semi-circular canals it is a question whether functioning end-organs remain in any part of the internal ear. Dr. Friesner said that as he recalled the matter, Boris did not examine the internal ear histologically after his experiment. Now, just as Uffenorde thought that the fistula test was still positive with what we now consider a totally destroyed labyrinth, so Boris may have gotten whatever action he did get from direct stimulation of the nerve endings. One should look with grave doubt on any conclusions regarding the physiology of any end-organ in the labyrinth where so large a part as the semi-circular canals was destroyed.

Dr. Fisher had emphasized perhaps one of the most important points, in that at times the functional tests are of exceedingly great clinical value. Dr. Friesner said that he himself happened to be associated with

neurologists of such excellence that he was not often able to help them, but that he had observed one thing: that where he found definite past-pointing, spontaneous past-pointing, etc., the neurologists find ataxia dysdiadokokinesis, with other signs, so that he did not often help them, but that they were occasionally able to localize a lesion in a hemisphere on the side opposite to that where the neurologists placed it. In one particular case, however, it was a bilateral lesion; however, most of the lesion was on the side where Dr. Maybaum placed it in accordance with functional tests which he made. The functional tests are far from useless, and should not be discredited. It was very much in the line of soiling our own nests if we do. One should keep an open mind in the matter, for there are many discrepancies which have not been explained.

Dr. Kopetsky had mentioned a case of specific neuritis in which the tests were bizarre; but Dr. Friesner had seen a similar case, which had improved under treatment. One of the earliest cases reported in Fisher's book was a specific neuritis in which the external semi-circular canal failed to react, etc.; so that while it is evident that there are many irregularities in the functional tests which still need explanation, the otologists, least of all, should be ready to discard them utterly.

DR. KERRISON said that Dr. Eagleton's and Dr. Kopetsky's papers were both of unusual interest. Both, however, were papers which required careful reading before they could be discussed with any real appreciation of their subject matter. Dr. Eagleton's paper, particularly, was one which it would be mere pretense to discuss without careful reading.

With regard to Dr. Kopetsky's paper, Dr. Kerrison said it was exceedingly useful to have the later views on labyrinthine physiology and reactions to disease and to different stimuli placed before otologists in such clear terms. It was also of decided practical value to have the more unusual vestibular reactions described.

Dr. Kerrison agreed with Dr. Fisher that nothing in the more recent studies of the labyrinth or in clinical observations of unusual or atypical cases should be allowed to disturb our confidence in what have come to be regarded as the cassical symptoms of a suppurative invasion of the labyrinth or of any acute form of inflammation of the membranous labyrinth, which constituted one of the most unvarying and dependable syndromes known to medicine.

Dr. Kerrison expressed his appreciation of both of the papers of the evening which made this meeting one of quite unusual interest.

DR. ISRAEL STRAUSS: Before discussing these two very excellent papers I want to ask two questions. First, Dr. Kopetsky cited a case of endothelioma of the frontal lobe in which the quick component was absent before the operation. I would like to know if he tested the man after the operation and if the quick component came back after the patient's recovery.

Second, will Dr. Eagleton tell us just what he means by "compensation" as a function of the cerebellum. I hope he will enlighten us on that point, for I don't quite understand just what he means by that term as applied to the cerebellum.

As a neurologist, and speaking for some of the neurologists in New York and for most of those in Philadelphia, perhaps some in Chicago and for a few in Boston, I feel that we owe a great deal to the otologists for the work they have done on the labyrinth and its connections with the cerebellum and cerebrum and the functions of those various portions of the central nervous system and the peripheral system. The neurologists did not for a long time pay much attention to this work, but as the years have gone by they have taken it up and have learned something from this study.

I will admit at once what Dr. Fisher says, and I know that Dr. Kopetsky will agree with me, that the diagnosis of tumor in the pontificial angle can be made much earlier by otologists using the vestibular tests than by the neurologists who depend on other neurological symptoms.

It is true that frequently, and even before we had the benefit of these tests, we, as neurologists, were able to diagnose tumors in various

locations, and even now, with these tests at our disposal, I would not say that in every case we could not make a diagnosis and dispense with these tests, but that is no argument either for or against the value of the tests. The point is that in medicine every test has its value, and every test and every symptom has to be taken into consideration in relation to other symptoms; so that the work the otologists have done in developing these tests in the conditions named have been of tremendous value to us.

I don't know but that Dr. Kopetsky and Dr. Fisher would agree regarding the loss of the reaction from past-pointing when testing the vertical canals, where we neurologists suspect intracranial pressure. We have learned to disregard this symptom as having any significance, so far as localization is concerned, for we find that the absence of that symptom is frequent in cases of intracranial pressure from any cause. However, in a case where we suspect intracranial pressure and are unable to find the cause for the condition, the absence of this reaction to caloric stimulation is again another symptom of significance and has to be taken into consideration. As neurologists, we cannot go into the finer details with Dr. Kopetsky and make the examination of variations that he is able to do, and I feel in his calling attention to these variations and pointing out to us all, irrespective of what branch of practice, that it is not fitting to be too dogmatic in our assertions nor to draw too fixed conclusions, his paper has done us service. I feel certain after reading his paper that it was far from his object to discourage us from using these tests or to deprecate in any way their value. As I see it, his object was merely to point out to us that a great deal is still unknown regarding their anatomical and physiological basis, and that therefore we should be a little cautious in being too certain of what we conclude.

Coming to the question of anatomical knowledge, we must not forget that a great deal is unknown regarding the connections in the brain, that we frequently postulate pathways that have not been satisfactorily demonstrated. None of you will have forgotten so much of your knowledge as not to know that the pyramidal tract fibers end in the cord, but probably few of you know that none of us have ever demonstrated connection fibers with these anterior horn cells, and yet we are convinced that there is such a connection, although it has not been demonstrated; and the same thing is probably true of other pathways.

Another thing to be remembered is that we as neurologists are getting away from enclosing functions too definitely, for we have learned that it is very hard to limit these cells into definite groups, and we are learning that different parts of the brain can assume the function of parts that are destroyed or injured. When you come to attempt to localize in the brain by experimental methods any function, you are running a great danger, because you are producing artificially often much more damage than you think or know about, and are in danger of injuring structures that you have no idea about, and your conclusions from loss of function may be wrong, and the conclusions drawn a short time after your experiment may also be foolish. If you had waited long enough you might have formed very different ones. This is especially true about experimental work on the cerebellum and, as Dr. Friesner has said, I would be very skeptical about much of the work that has been done in that direction. Probably much of the lack of agreement by the different experimenters is due to the methods they have used, thus causing differing results.

A word about past-pointing. I think Dr. Kopetsky meant to convey the idea that the attempt to localize in any lobe or lobule of the cerebellum a loss of function in any limb or pair of limbs by lack of past-pointing is trying to do more than you are able to do, and that the attempt by Barany and some others to use these tests in trying to be too exact in their localization has probably made some people become more or less skeptical about the general value of the work.

DR. EMIL ALTMAN confirmed what Dr. Kopetsky had said about the case of frontal lobe tumor with absence of fast component. Dr. Altman

had asked Dr. Strauss to see the case with him and he also thought that it might be a case of cerebellar tumor, but thought it was a frontal lobe tumor. The case was watched for several weeks and proved to be a frontal lobe tumor. The man is still under Dr. Altman's care and further tests will be made. The case is not finished. It will be reported in full at some future time.

DR. RALPH ALMOUR: The trend of the discussion was toward a general misinterpretation of the purpose of our paper. I want to correct this impression at once. We have not attempted, nor ever will attempt, the discouragement of the use, clinically, of so valuable a diagnostic aid as the labyrinth tests. But, when a group of scientists are content to leave unexplained, a series of reactions and phenomena, the knowledge of which is still in its infancy, then they are not loyal to the best traditions of medicine. No progress was ever made in medicine except by an intense study and a minute investigation of everything pertaining to it. No progress was ever made except by a thorough understanding of the fundamentals of medicine, and it is upon these that we build, and continue to build, until we shall have obtained a mighty, infallible structure. And so, when otologists are satisfied to call "quits" and continue blindly with the use of the tests, getting a certain percentage of positive and negative results, it is time that they were brought to the realization that these tests could be made far more accurate and valuable as diagnostic measures were their fundamental physiology and mechanics more thoroughly understood.

We are well aware of the large number of cases of intracranial lesions that have been diagnosed by means of the labyrinth tests. We feel, however, that these cases can be diagnosed more accurately from the other neurological signs that they present, and that the labyrinth tests should be considered as confirmatory evidence rather than as the diagnostic evidence. We are also well aware of the large number of times the tests have failed to establish a diagnosis of intracranial disease where it was present, and should have given definite deviations from the normal. I recall one instance, a case reported before this Section by Dr. Rosenbluth, wherein a tumor of both cerebellar hemispheres, invading the inferior peduncle, was found at autopsy. The findings on repeated labyrinth tests were all normal. It is with the idea of stimulating further investigation, so that we may be able to reach a more definite explanation of the phenomena elicited by testing the labyrinth, and so arrive at a logical basis for the interpretation of our failures, that this paper was presented. Dr. Kerrison has summed up the situation when he said that the paper was presented to stimulate thought.

Dr. Fisher has stated that he cannot help but feel that the caloric reactions and the rotation reactions emanate from the same part of the labyrinth. As an example, he cites the finding of the absence of the quick component, and a resulting conjugate deviation of the eyes, following both caloric and rotary stimulation, and concludes that this phenomenon leads him to believe that the reactions are produced by the stimulation of the same part of the labyrinth. We know, however, that the quick component is not vestibular in origin, but, on the contrary, is distinctly a compensatory action on the part of some portion of the cerebrum. Because it is present following both caloric and rotary stimulation, it cannot be used as a basis for the determination that both reactions originate in the same part of the labyrinth.

In answer to Dr. Strauss, we have not again tested the man, and realize the importance of doing so. I agree with Dr. Strauss, that primary eighth nerve tumors, which include most of the cerebello-pontine-angle tumors, can be diagnosed by the labyrinth tests alone. It is here that these tests have their greatest clinical value. The other intracranial neoplasms will only produce responses when they interfere with the retro-labyrinthine pathways, but, owing to the vagueness which surrounds the theoretical knowledge of these supposed pathways, we are not justified in continuing to diagnose definitely the location of these lesions from the disturbances in the labyrinth reactions alone when they

present no other neurological symptoms, and give no spontaneous phenomena. Rather is it to be hoped, that, with the further use of the tests, and with a careful investigation of the fundamentals involved in the production of the reactions, we may reach a stage where we shall be really able to do that which we are now fallaciously attempting to do.

DR. EAGLETON: You all missed the point of the paper: I am a firm believer in the value of the vestibular reactions. They are of the greatest value. We know now that a lot of the postulates that have been laid down are not true. The vestibular reactions are like the clinical thermometer, not like the Wassermann test. We use the turning chair and cold caloric and it frequently shows something wrong within the cranium. It, however, is of localizing value but infrequently and that in acute lesions. If you take the temperature of a patient and he has fever, you do not say that he has typhoid fever, but it gives a valuable clue, and so with the vestibular reactions they make up a part of the neurological picture.

The reason why the tests are said to be confusing by Dr. Kopetsky is because we have not the clinical basis on which to interpret the abnormal findings. My paper called attention to the fact that in acute lesion we may have definite reactions, but they do not last, whether the lesion is in the cerebellum or elsewhere. If a man receives an injury to his cerebellum, he has certain definite reactions that will last three days. If you injure the tempero sphenoidal lobe, he has a loss of the quick component for some few hours but it also rapidly disappears. If you have a cerebellar lesion by a gunshot wound, you have a definite and distinct nystagmus. It is different from any other nystagmus. This nystagmus is not to the right or left, but from "a rest point", but it disappears in a few days. These are clinical facts.

Now, we should study the reactions in our tumors, our abscesses, and take the history in its relation with these vestibular reactions. We should ask ourselves: Did this come on yesterday or has it been a slow-growing lesion? We can but infrequently obtain localizing value from the vestibular reactions. But we can diagnose that the patient has increased intracranial pressure of the posterior fossa from the vestibular reactions. I stated this in 1912 as a new symptom of intracranial pressure.

We can also tell that the meshes of the piarachnoid have been invaded extensively by a suppurative or hemorrhagic process if we turn a patient from this way and then that way, and find that he has a reduction in the direction of the nystagmus (of 50 per cent of the normal in apparently healthy individuals). If the insurance company says there is nothing the matter with the man, the vestibular findings are definite and last a long time, but other deviations disappear, and rapidly disappear, I say by compensation. But they do disappear and the only way to make use of them is to use them as a thermometer. The thermometer shows the temperature, the history and the other findings may find the cause. If we can only get enough cases of post-mortem findings and compare them with the previous history, and ascertain how long they have lasted, we will have something that will help us.

DR. DOPETSKY: It matters little who wrote Homer's poetry. It is of less concern whether Barany first called attention to an observation or whether Borries did. It does matter, however, whether the observation is substantiated by subsequent experimental and clinical study of cases. It very much matters that this later work offers a more logical explanation of findings which are obtained at the bedside and permits an interpretation which more often than without it makes for a clinical diagnosis.

Dr. Friesner criticizes the experiments of Borries. If what Friesner says of them is true, what has he to say to the crudities and questionable methods of the experiments which were originally made and upon which our whole theory of reactions of the labyrinth is based and which have been accepted as deducing facts and which have been copied from text-book to text-book without scrutiny or question.

Dr. Fisher gave the discussion a turn that was not intended. Our paper was not meant to disparage the labyrinthine tests. In fact, we urged that the tests always be made—and that, wherever possible, necropsy studies be performed to endeavor to find the reasons for the reactions obtained.

We have a peculiar situation! One group of observers accept the results of experimentation which is itself open to question; and when the reactions from the clinic do not coincide with the laboratory they designate their results "Paradox Reactions". When we attempt further study of these so-called paradoxes and offer explanations of the contradictory findings, another group of observers arises and protests against the criticism of accepted postulates. To bring the newer work of authoritative observers to your attention and give the results of over two years of study of these problems, was the purpose of this paper.

I am in absolute accord with what was said by Dr. Strauss and Dr. Friesner; and the most gratifying part of this interesting evening is that Dr. Eagleton, working from an altogether different angle, should arrive at almost similar conclusions to mine.

I would sugest to those who think we would discourage the use of the labyrinthine tests that they read the paper carefully when it is published and they will see that I neither say nor suggest anything of the kind; but I do say that too much stress has been laid by *some otologists* on the diagnosis of conditions by this method where neurologists could more easily diagnose them without the tests being made at all. The tests are valuable corroborative evidence in tumors. In suppurative labyrinthine diseases they have a distinct value. Study and record observations, and we will make progress. It is no sign of progress to repeat, and repeat again, statements based on questionable experimentation. We shall be able to make more progress if we accept Borries' statements as a new step in the development of diagnosis. I yield to no one in my respect for the work of Fisher and Jones as far as that work goes; but we cannot stop at this point.

Dr. Eagleton's paper showed intense and original thought. Sometimes we may question his deductions and the logic by which he approaches his conclusions, but even that is preferable to a quiet acceptance of the thing that is stated and restated. He, too, makes conclusions that are intensely interesting to me; and far from throwing a mantle of gloom over the labyrinthine tests, I feel stimulated to think that from another angle there is evidence that we are tending toward a better understanding of the tests and are arriving at a new evaluation of them as an aid toward intracranial diagnoses.

On a meeting held on February 6, 1924, the Danish Oto-Laryngological Society has adopted the following resolution:

On November 1, 1922, the Society resolved to convoke an international congress of otolaryngologists, to be held in Copenhagen in 1925 or 1926. In view of this resolution having been made under the supposition that the congress should be strictly international, and invitations be issued to all nations, and in view, further, of the fact that, according to the firm conviction of the Society, no conditions be at hand pro tem, for an international congress to be held, it has now been decided to postpone the invitation to the said congress till a point of time more suitable for the realization of the plans than the present.

For the Committee of the Danish Oto-Laryngological Society,
Copenhagen. JENS KRAGH, *Secretary.*

Weed Hay Fever

THE Late Summer and Early Autumn Type of Hay Fever is caused by many of the common weeds, as-Ragweed, Russian Thistle, Sage Brush-the species differing widely according to locality. To assure specific treatment diagnostic tests are essential. To assure preseasonal treatment early diagnoses should be made, otherwise the less desirable, though often beneficial, coseasonal treatment will be involved. List of late flowering weeds, showing regional distribution and time of pollination, sent on request.

GIANT RAGWEED *Ambrosia trifida*

RUSSIAN THISTLE *Salsola pestifer*

SAGE BRUSH *Artemisia tridentata*

The Arlington Chemical Company

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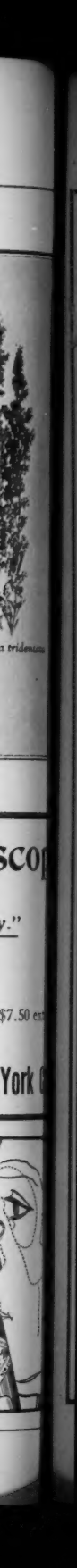
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